SURGERY, GYNECOLOGY AND OBSTETRICS

AN INTERNATIONAL MAGAZINE PUBLISHED MONTHLY

VOLUME XLI OCTOBER, 1925 NUMBER 4

TERATOMATA—OVARIAN AND RETROPERITONEAL[1]

BY ONSLOW A. GORDON, JR., M.D., F.A.C.S., BROOKLYN
Clinical Professor Gynecology, New York University and Bellevue Hospital; Obstetrician, Carson C. Peck Memorial Hospital

THE classification of ovarian neoplasms is at the present time a subject upon which there is a marked lack of unanimity of opinion. That this is to some degree unavoidable is apparent when one considers the multiplicity of growths both cystic and solid arising from the ovary. The need of a more uniformly accepted terminology is appreciated when we consider the question of ovarian teratomata. We find the terms teratoma, embryoma, teratoid, teratomatous, dermoid, and mixed cell tumor used at times interchangeably.

Lynch (15) uses the term embryoma and states that this includes dermoid and teratoma, the dermoid being essentially benign and the teratoma malignant. Bland-Sutton (3) follows much the same classification considering teratomata and dermoids as specific entities. Strong (19) refers to genital and extragenital teratomata. Graves (11) considers dermoids and teratomata separately, at the same time referring to a similar histogenetic origin. Ewing (6), Frank (7), Frankl (9) and others refer to ovarian teratomata as of two types, cystic and solid. The cystic is the more common, orderly arranged dermoid; and the solid, the unusual potpourri of tissue found in the malignant teratoma. This classification seems to me to be preferable from the standpoint of histogenesis and clarity. The common, benign, cystic teratoma or dermoid, and the rare and malignant solid teratoma are probably identical in origin and there are many intermediate types connecting the two. As to retroperitoneal teratomata Kolb (12) states that no solid retroperitoneal teratoma has been reported. A careful survey of the literature to the present time substantiates this statement.

Ovarian dermoids or cystic teratomata in any location are to be carefully distinguished from true dermoids which are congenital sequestration tumors found at the lines of embryonic fusion. These tumors arise by the displacement and inclusion of ectodermal cells.

The histogenesis of ovarian teratomata is interesting but highly speculative. Any speculation as to the origin of teratomata must accept the fact that the cell from which they originate is totipotent as the growth shows evidence of all three primary germ layers, ectoderm, entoderm, and mesoderm. There are at present two seriously considered theories as to their origin; one the so-called blastomere theory of Marchand and Bonnet (4) and the other the more commonly accepted germ cell theory of Wilms (21). The blastomere theory assumes that the histogenesis of these tumors dates from the earliest segmentation of the fertilized ovum, for it is at this time that the blastomere originates. In some way a blastomere becomes isolated persisting as a quiescent parasite upon its host, or fetus *in fetu*, from the time of earliest seg-

[1] From the Laboratory of the Carson C. Peck Memorial Hospital. Read before the Brooklyn Gynecological Society March 6, 1925.

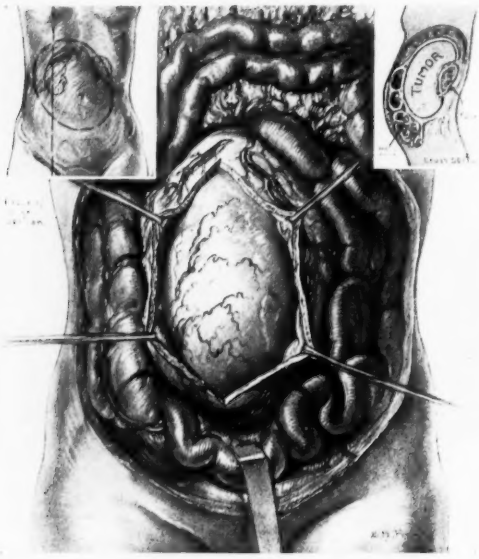


Fig. 3. Retroperitoneal teratoma *in situ*.

Fig. 4. Retroperitoneal teratoma after removal showing the right kidney embedded in its capsule. Specimen measured 32 by 41 centimeters.

mentation through fetal and into postnatal life; when activated by some unknown stimulus it gives rise to a teratoma. This theory readily accounts for the unusual extra-ovarian teratomata found in the skull, anterior mediastinum, or retroperitoneal tissues. The more commonly accepted theory is that teratomata originate from germ cells; whether this growth is called parthenogenetic or not is immaterial. By this explanation alone can we account for the marked predilection of teratomata for the sex glands. The multiple origin of cystic teratomata or dermoids in the ovary can hardly be assumed to arise from multiple isolated blastomeres. Novak (16) reports ten dermoids in one ovary and eleven in the other. Cases of this type give great weight to the germ cell theory of origin. On the other hand we cannot account for the occasional teratoma of the mediastinum or pineal gland by this theory. Thus there are valid objections to both the blastomere and the germ cell theory.

Retroperitoneal teratomata may originate as do ovarian teratomata, from an isolated blastomere or from germ cells of an accessory ovary retroperitoneal in location. They may originate from germ cells which have remained at their primary embryonic location, retroperitoneal and lateral to the spinal column. This explanation seems to apply best to the case I wish to report.

Cystic ovarian teratomata or dermoids are one of the commonest ovarian neoplasms. Their frequency is variously estimated at from 2 to 18 per cent of all ovarian tumors: Spencer Wells finds 2.2 per cent in a series of 1,000 ovarian tumors; Olshausen 4 per cent in something over 2,000 ovarian tumors; Bishop (2) in a recent review gives 7 per cent in 333 cases occurring at The Brooklyn Hospital; Kelly 18 per cent in 138 cases. During the past 5 years there have occurred on the gynecological service at Bellevue Hospital approximately 125 ovarian tumors with 10 cystic teratomata, 8 per cent.

Bilateral cystic teratomata are comparatively common. Olshausen found 14 per cent to be bilateral. Pfannenstiel states that they occur in 10 per cent of cases. Gebhard

Fig. 5.

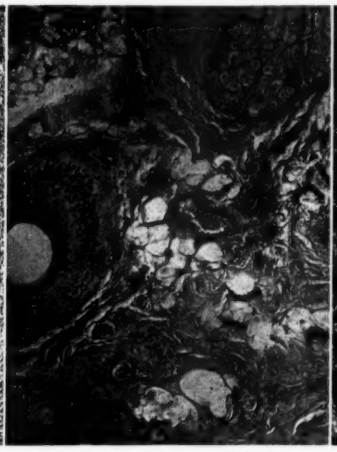


Fig. 6.

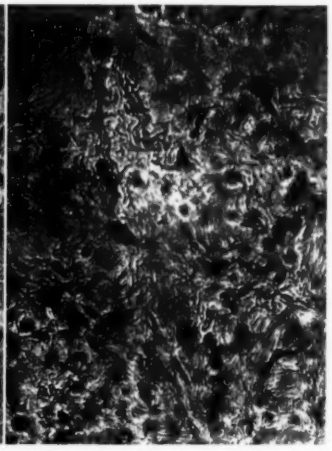


Fig. 7.

Fig. 5. Section from retroperitoneal teratoma showing one of the many cysts lined by a palisaded layer of epithelium.

Fig. 6. Section from retroperitoneal teratoma showing skin structures and hair follicle.

Fig. 7. Section from retroperitoneal teratoma showing an area of myxomatous connective tissue with peculiar cells designated as "bird's eye" cells. They are neoplastic epithelia scattered here and there in various sections throughout the tumor.

in 107 cases found 16 bilateral growths. Kelly found one in 87 cases, and Weiner one in 60 cases.

Malignant changes of any type in cystic teratomata are rare. The most frequent type of malignancy is carcinoma. This is usually of the squamous cell type and arises from the extensive epiblastic elements of the tumor. The ratio of development of carcinoma is usually given as occurring in from 3 to 5 per cent of the cases. Frankl (10) in 1920 was, however, able to collect only 60 authentic cases. Sarcomatous change in a cystic teratoma is very rare, and but a few isolated cases have been recorded. These figures would seem to justify conservation of a portion of the ovary or ovarian resection in cases of bilateral cystic teratomata occurring in young women in whom subsequent pregnancies are greatly desired.

Solid teratomata are among the rarest of ovarian neoplasms. They are characterized by embryonic tissue derived from all three layers of blastoderm. The tissue is in a confused association without the attempt at definite structure so manifest in the cystic teratoma. The solid teratomata develop rapidly and are highly malignant. Graves states that there are less than 50 cases of solid ovarian teratomata in the literature at the present time. In 1907 Frank (8) was able to collect only 37 authentic cases. Kroenig (13) reports 40 cases from the literature up to 1919. There is no case of solid teratoma in the records of Bellevue Hospital where the gynecological admissions will average 1,800 cases a year. It has been suggested that the solid teratomata consisting largely of embryonic tissue originate from immature sex cells while the cystic teratomata consisting largely of adult tissue originate from more mature sex cells. Askanzy (1) assumes that the adult tissues of the dermoid are of equal age with those of the host and that the tumor is in fact congenital, while the solid teratomata showing embryonic tissue are postnatal in origin. Rossle (18) reports the case of a well developed dermoid in an infant of 10 months. Lexer (14) reports a cystic teratoma in a female child of 7 weeks. Eggenberger (5) reports a dermoid the size of a child's head in an 8-year-old child. Polak (17) states that prior to puberty dermoids are the commonest ovarian tumors.

The so-called struma ovarii in which evidence of thyroid tissue is found represent a

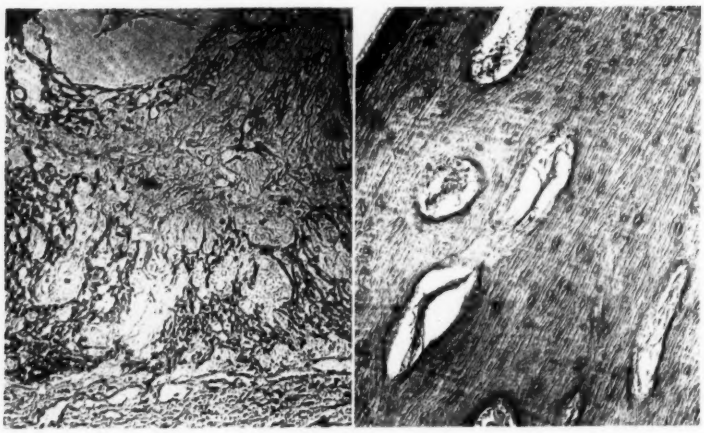


Fig. 8 (left). Section from retroperitoneal teratoma showing a disorderly invasion of loose connective tissue and myxomatous tissue by epithelia. A potentially malignant area.

Fig. 9. Section from retroperitoneal teratoma taken through plaques of bone showing normal bone formation.

one-sided growth in a teratoma. They may be benign or malignant. Pick, in 21 dermoids, found islands of thyroid tissue.

Retroperitoneal teratomata are as a matter of course decidedly unusual tumors. Their etiology, namely, that they probably arise from germ cells of the ovary or isolated blastomeres makes this location unusual. Lexer and Kolb have studied retroperitoneal teratomata most thoroughly. Lexer, in a monograph on abdominal teratoid tumors, both intraperitoneal and retroperitoneal, reports four retroperitoneal teratomata. He states that most retroperitoneal teratomata are found in the left retroperitoneal tissue near the vertebral column. Kolb, writing in 1909, states that Tilloux (20) in 1886 collected 33 cases of extraovarian abdominal teratoid tumors; these were intraperitoneal and retroperitoneal. Subsequent to this time the literature upon the subject has been sparse, especially references to retroperitoneal teratomata. Kolb collected five cases from the literature and added one case of his own.

The retroperitoneal teratomata are usually cystic and well encapsulated. They produce symptoms chiefly by pressure. There is no case on record of a solid or malignant tumor of this type.

CASE REPORT

The case I wish to report occurred in a woman 50 years of age. She was admitted to the Peck Memorial Hospital under the care of the writer October 5, 1924. She complained of abdominal pressure symptoms and an increase in the size of her abdomen for about 3 months prior to her admission to the hospital. She had no menstrual disturbance, having passed through a normal menopause 5 years prior to this time. Abdominal examination showed a plainly palpable tumor mass apparently cystic in character occupying the larger part of the abdomen. A pre-operative diagnosis of large ovarian cyst was made. She was operated upon on October 8, 1924. Operation revealed a negative pelvis, the uterus, both tubes, and ovaries being normal. It was apparent at once that the tumor was retroperitoneal as the posterior parietal peritoneum was almost in apposition with the anterior parietal peritoneum at about the umbilicus. The posterior peritoneal layer was incised and by blunt dissection the tumor enucleated. The tumor apparently arose from the cellular tissue to the right of the vertebral column although there was no definite pedicle. The right kidney was adherent to the wall of the tumor and during attempted separation the kidney was traumatized; it was, therefore, removed with the tumor mass. The patient made an uneventful recovery, leaving the hospital 16 days after operation.

For the detailed histological study of this tumor I am indebted to Dr. Hala, pathologist to the hospital, and I will quote from his report: "Specimen consists of a cystic mass 41 by 32 centimeters, weighing 11 kilograms (26 pounds). It possesses a thick wall which superficially contains a marked

amount of adipose tissue. Internally it is trabeculated and shows numerous small and large subsidiary cysts filled with brownish gelatinoid material. Occasional plates of what appears to be osseous tissue are found embedded within the wall. The contents of the main cystic cavity consists of a semigelatinous brownish fluid. The specimen of kidney shows no gross pathology. Sections were taken from various portions of the tumor, including what appears to be plaques of bone. The microscopic picture is variable. Necrosis is a prominent feature. In general the structure is of fibrous character. Scattered here and there are circumscribed collections of epithelial cells forming here acini, and in other places solid alveoli. Some of the acini are lined with a palisaded layer of epithelium. In still other areas the epithelial cells are diffusely scattered throughout an œdematous fibro-adipose stroma in a disorderly fashion. Such areas suggest a potential malignancy. In the various sections one encounters areas of what appear to be true myxomatous tissue. Histological examination of the sections apparently incorporating plaques of bone disclosed a fibrillar connective-tissue stroma in which small areas of histologically normal osseous tissue are present."

While in this tumor we found positive evidence of only two primary germ layers, ectoderm and mesoderm, I feel that the tumor should be designated as a teratoma rather than a teratoid or mixed cell tumor. Only by a very thorough study can we determine the absence of entoderm. Ewing states that there is little doubt that entoderm is the least vigorous of the three germ layers, and may succumb early in the tumor growth.

SUMMARY

1. The term dermoid as applied to ovarian neoplasms is inaccurate. So-called ovarian dermoids should be referred to as cystic ovarian teratomata.

2. The histogenesis of ovarian teratomata is highly speculative. There are two seriously considered theories as to their origin, one the so-called blastomere theory of Marchand and Bonnet, and the other the germ cell theory of Wilms.

3. Cystic teratomata or dermoids are one of the commonest ovarian neoplasms. In a series of 125 ovarian tumors at Bellevue Hospital they comprise 8 per cent of the cases.

4. Bilateral cystic teratomata are comparatively common, occurring in from 2 to 14 per cent of the cases.

5. Malignant changes in cystic teratomata are rare, the most frequent type being squamous cell carcinoma. The ratio of development of carcinoma is usually given as from 3 to 5 per cent.

6. Ovarian resection is justifiable in cases of bilateral cystic teratomata in young women in whom subsequent pregnancies are desired.

7. Solid teratomata are among the rarest of ovarian neoplasms. There is no case in the records of Bellevue Hospital in which the gynecological admissions average 1,800 cases a year.

8. So-called struma ovarii may be benign or malignant.

9. Retroperitoneal teratomata are unusual tumors. There is no case of solid retroperitoneal teratoma recorded in the literature.

10. Report of a case of cystic retroperitoneal teratoma weighing 11 kilograms (26 pounds).

REFERENCES

1. Askanzy, D. Verhandl. d. deutsch. path. Gesellsch. Dresden, 1907.
2. Bishop, E. Am. J. Obst. & Gynec., 1924, vii, 3.
3. Bland-Sutton, J. Keen's Surgery, vol. 1, p. 818.
4. Bonnet, R. Ergebnisse de Anatomie und Entwickelungsgeschichte, 1899, p. 820.
5. Eggenberger. Baseler med. Gesellsch., 1908, Feb.
6. Ewing, J. Neoplastic Diseases, 1st ed., 1919, p. 593.
7. Frank, R. T. Gynecological and Obstetrical Pathology; Gynecological and Obstetrical Monographs, 1922, p. 417.
8. Idem. Am. J. Obst., 1907, lv, 3.
9. Frankl, O. Handbuch der Frauenheilkunde, 1914, p. 216.
10. Idem. Centralbl. f. Gynaek., 1920, xliv, 313.
11. Graves, W. P. Gynecology, 3rd ed., 1923, p. 428.
12. Kolb, K. Das retroperitoneal Teratom. Heidelberg, 1909.
13. Kroenig, O. Lehrbuch der Gynaekologie, 1919.
14. Lexer, E. Arch. f. klin. chir., lxi, 62.
15. Lynch, F. W. Pelvic Neoplasms, Gynecological and Obstetrical Monographs, 1922, p. 356.
16. Novak, E. Beitr. z. path. Anat., xlv.
17. Polak, J. Manual of Gynecology, 2nd ed., 1922, p. 344.
18. Rossle, C. Centralbl. f. Gynaek., 1912, p. 36.
19. Strong, L. W. Am. J. Obst., 1918, lxxviii, 5.
20. Tilloux. Del airrier cavite des epiploons. Gaz. d. hôp., 1886, p. 757.
21. Wilms, V. Arch. f. Gynaek., 1900, lxi, 203.

THE CALCAREOUS DEPOSITS OF SO-CALLED CALCIFYING SUBACROMIAL BURSITIS[1]

By JOHN BERTON CARNETT, M.D., Philadelphia, Pennsylvania
Professor of Surgery, University of Pennsylvania Graduate School of Medicine

I HAVE personally operated on 14 cases of the condition commonly called calcifying subacromial bursitis for the removal of calcareous deposits, and have assisted my former Chief, Dr. Edward Martin, in operation on 5 other similar cases. I have seen more than 25 shoulders in which a calcareous deposit was demonstrated by the X-ray but in which no operation was performed. In many of the operative cases there were bilateral deposits but operation was performed on only one shoulder.

Anyone attempting to familiarize himself with the literature dealing with these cases should read the papers of Codman in which he describes subacromial bursitis as the most common cause of stiff and painful shoulders. I believe that further investigation will demonstrate that a variety of lesions, of which calcareous deposit is one, may give rise to the symptom complex now usually ascribed to subacromial bursitis. Codman's contributions are classics in dealing with the mechanism of normal shoulder movements. His observations are supplemented by those of Stevens, who has studied the action of the short rotators of the shoulder. Codman's clean cut description of the symptoms of subacromial bursitis and the mechanism by which they are produced has been accepted without much alteration by subsequent writers. I endorse the majority of Codman's observations and will quote freely from them in this paper.

Calcareous deposits were unknown when Codman published his first paper in 1906 and he makes no mention of them in it. Painter in 1907 was the first to report the finding of definite shadows in the shoulder by X-ray examination in 4 cases. He assumed that the shadows were caused by thickening of the walls of the subdeltoid bursa as did Baer, who also reported 2 operative cases in 1907. In the light of our present knowledge there can be no doubt that both these authors were dealing with calcareous deposits. They each reported having excised the entire subdeltoid bursa in 4 cases. Painter found the deposit in 2 of the specimens removed at operation but missed the deposit in the other 2 probably because he did not search for it underneath the floor of the bursa.

Painter and Baer reported the deposits as being located within the cavity or walls of the bursa itself but Codman saw Painter operate on one of the latter's 4 patients and observed that the deposit was not in the bursa but was located beneath the floor of the bursa. Codman reports Painter as agreeing with him that the deposits were beneath the bursal floor rather than in the bursa. Codman reported one case of his own in which the deposit was found at operation beneath the floor of the bursa. Codman was, therefore, the first to note the extrabursal position of these deposits. Many subsequent writers in describing single operative cases report the deposit as being in the bursa but it seems highly probable that the great majority of them did not make exact observations as to the actual position of the deposit in relation to the bursa. Their writings contain many other fallacies which still permeate recent literature notwithstanding Brickner's forcible efforts to overcome them in his two excellent papers in which he reports 18 operative cases.

The literature is confusing also because various writers use the terms "subdeltoid bursa" and "subacromial bursa" as though they were synonymous and interchangeable, whereas other writers believe these two terms represent two separate and distinct bursæ. The descriptions contained in different books on anatomy are likewise variable and confusing. My own experience indicates that Piersol's *Anatomy* and Codman's and Brickner's papers are correct in describing only one bursa which lies in part beneath the acromion process and in part beneath the deltoid muscle.

[1]Read before College of Physicians of Philadelphia on October 1, 1924.

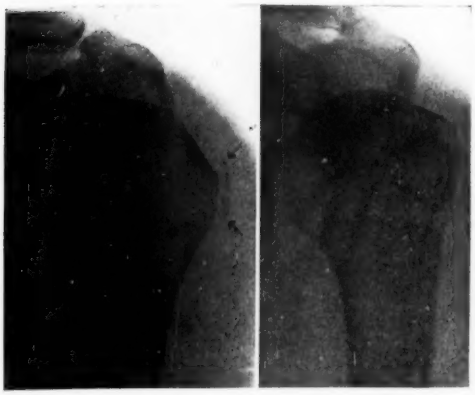


Fig. 1. *A* (left). Internal rotation. Large shadow 12 hours after onset of first symptom. *B*. External rotation. Deposit concealed.

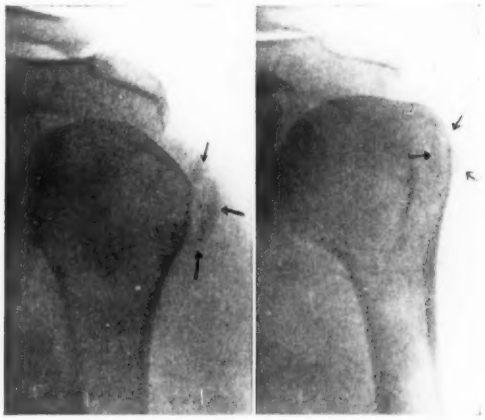


Fig. 2. *A* (left). Internal rotation. Large shadow, shown free from bone. *B*. External rotation. Deposit partly hidden, simulating an osteophyte.

Surgical literature contains detailed histories of numerous cases quite similar to those I have encountered and it seems needless to give the individual records in each of mine. The clinical histories suggested a division of these patients into two main groups, (1) the acute and (2) the chronic.

The outstanding symptoms in the acute cases were pain and fixation of the shoulder joint. The maximum pain was located commonly at the outer side of the arm over the lower half of the deltoid muscle and only exceptionally at the site of the calcareous deposit just beneath the acromion process. In many of the cases, especially in those of longer duration, the pain was referred from the neck all the way to the hand. Many of these cases had been undergoing treatment for brachial neuritis for several months. Several of them were tender along brachial nerve trunks but none of them had reactions of nerve degeneration. In all of the acute cases pain was aggravated by active and passive motions of the shoulder, particularly in the direction of abduction and inward rotation. The pain was usually worse at night apparently due to the backward drag of the elbow. The patients could not lie on the affected side because direct pressure aggravated their pains. Many patients had learned they could obtain some relief at night by placing the arm on a pillow in a position of slight abduction. The pain varied in intensity in different patients and in the same patient on different days. In some the pain was intolerable except when relieved by full doses of opiates. In the hyperacute cases the pain was as intense as that encountered in renal or biliary colic. In the non-operative cases there was a natural tendency for the hyperacute pain to ease up after a few days or within two weeks. On partial subsidence of the pain patients were prone to resume active use of the arm too early and pain recurred. Usually the pain became less severe and all the symptoms merged over into the chronic type of the disease.

In the acute cases the range of motion at the shoulder joint was greatly restricted by muscle spasm and pain. There was never complete fixation nor ankylosis of the joint. The shoulder was least restricted in backward and forward swinging motions at the side of the body. After the patient's confidence had been gained passive abduction of the arm was usually free through an arc of about 10 degrees directly outward from the side of the body. Further abduction, either actively or passively, was not possible in the shoulder joint itself but by rotating the scapula on the chest nearly all these patients were able to bring the arm outward to about a 40 degree angle from the lateral chest wall.

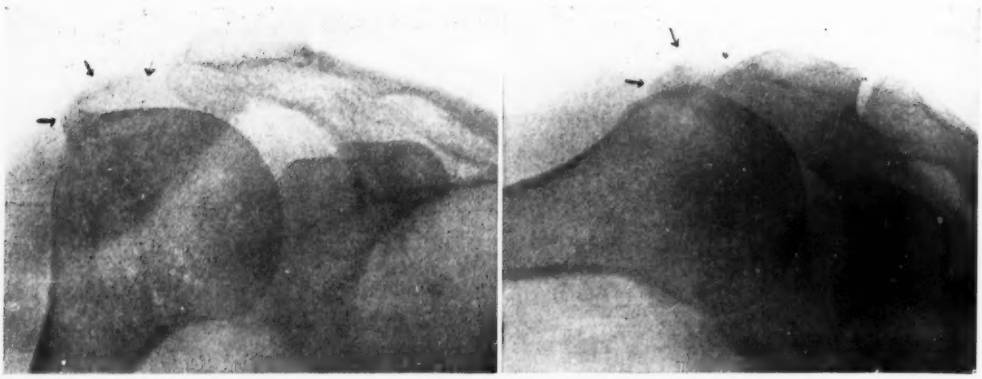


Fig. 3. *A* (left). Arm in adduction. Large deposit. *B*. Arm in maximum possible abduction (about 85 degrees). Deposit apparently impinging on acromion.

Inward rotation was restricted in all acute cases as evidenced by an inability to place the hand behind the back. The patients were unable to comb the back hair and could not fasten suspender or rear skirt buttons. The men could not introduce the hand into the hip pocket. External rotation usually was fairly free but was restricted and painful in several.

In a few of the acute cases, atrophy of the deltoid, supraspinatus, and infraspinatus muscles was more marked than would be expected simply from non-use. In none of these patients was there a swelling or fluctuation indicative of fluid accumulation in the subacromial bursa. In 6 cases, efforts to obtain fluid from the bursa by aspiration proved futile. One patient only had œdema which was moderate in amount and was located in the lower deltoid region. The same patient was the only one to exhibit any fever and her temperature barely crossed 100 degrees. Skiagrams demonstrated the deposit of lime salts in every acute case.

The chronic type of cases either began as such or were originally of the acute type. Pain, both as to localization and radiation, was similar to the acute cases but varied greatly in intensity in the individual patients. In different patients the pain was constant, intermittent or remittent over a period of weeks, months, or years. Mild exacerbations of pain, lasting from several days to a few weeks, were frequent either as the result of exercise or without obvious cause. Several patients had very acute exacerbations in which the pain was as viciously severe as in the acute type of cases. In the milder cases and during remissions in the severer cases pain was felt only during extreme inward rotation or abduction to or above the shoulder level.

In elevating the arm from the side of the body to a perpendicular, some of the patients would experience pain only when the arm was passing through that portion of the arc from 75 to 90 degrees and on dropping the arm to the side would experience similar pain when the arm was within the same arc. A few of these patients could go through the same motion painlessly by holding the humerus in a position of external rotation (palm up and elbow slightly flexed).

Limitation of motion varied greatly in the chronic cases. In some, motion was practically free except for causing bearable pain in abduction; in extreme inward rotation; and in circumduction at the shoulder level. In the majority, however, there was more or less marked restriction in abduction and inward rotation either with or without restriction of external rotation. Limitations of motion were more marked during exacerbations of pain due to the pain and muscle spasm. Maintenance of the arm in its restricted position in long standing cases led to contractures of the shoulder muscles and to atrophy which was most evident in the deltoid, supraspinatus,

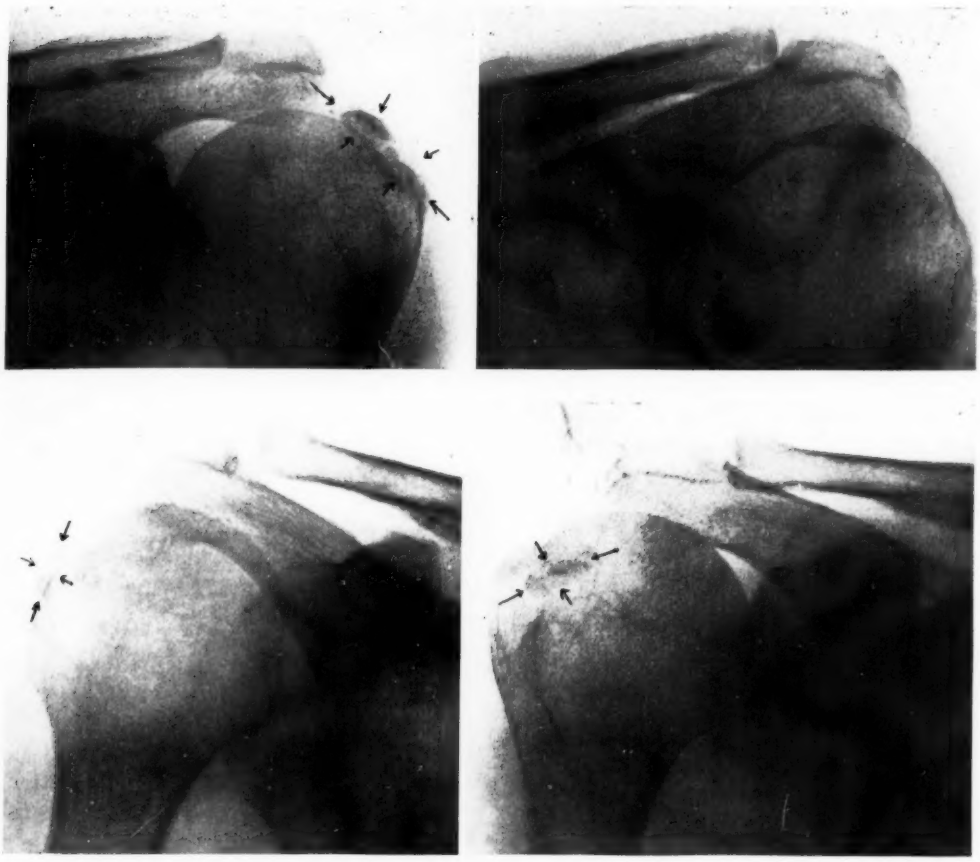


Fig. 4. Bilateral deposits. *A* (upper left). Left shoulder in external rotation showing multiple deposits which in *B* (upper right), are concealed in internal rotation. *C* (lower left). Symptomless right arm in internal rotation. The deposit shadow overlaps the bone shadow and resembles a bony outgrowth but stereoscopically the deposit shadow is free from the bone. *D* (lower right). Right arm in external rotation. Humerus and deposit shadows superimposed. No change in right shoulder shadow for 6 months. *E*. Deposit entirely gone.

and infraspinatus muscles. In a few of these patients the chief complaint was disability rather than pain.

In many chronic cases the condition apparently ran a self-limiting course and irrespective of treatment the patient made a complete recovery at varying periods from a few months up to three years. In a very small percentage of cases the condition persisted over a longer period of years (up to twenty years) and there were apt to be long periods of remission of symptoms. The calcareous deposit was demonstrated by a skiagraphic examination in every chronic case.

With but one exception, in all cases, both operative and non-operative, acute and chronic, the patients who were suffering either from pain or limitation of shoulder motion had a characteristic area of localized tenderness just beneath the acromion process on the anterior or anterolateral aspect of the upper arm, usually over the site of the lesser tuberosity. One operative case of the chronic type did not exhibit any localized tenderness

Fig. 5. Huge deposit on January 18, 1922. Spontaneous absorption. Shadows entirely absent on April 13, 1922, and September 27, 1924.

weeks and 2 weeks respectively prior to the acute exacerbation but there was exquisite tenderness the day of operation. The presence of a sharply localized finger-tip to quarter-dollar sized area of tenderness was found to be a valuable sign in making a differential diagnosis from a general arthritis of the shoulder joint as in the latter condition the head of the humerus is tender to finger pressure throughout its entire circumference. The majority of the patients, however, were not aware of having this area of tenderness until it was revealed to them by the surgeon's examination. Many patients were slightly tender over a corresponding area in the opposite and otherwise apparently normal shoulder. In several patients with unilateral symptoms but with bilateral calcareous deposits tenderness was absent in the shoulder that was free from pain and stiffness. The area of tenderness disappeared under the acromion in wide abduction (Dawbarn's sign) in all cases in which it was tested.

Nearly all of the 19 operative cases were either of the acute type or were in the acute exacerbation stage of the chronic type at the time they first came under observation and almost all of them were operated upon within the first 24 hours thereafter. The first one of these cases, a physician, was operated upon in 1907 by Dr. Martin. In the first 8 of the 19 operative cases, the sole aim of the operator was to remove the calcareous deposit and no effort was made to determine either the exac location of the deposit or the presence of possible pathology in the bursa itself. The deposit was removed in every instance and all but one untraced patient are known to have been cured. The conditions encountered in the operative field were studied very carefully in the subsequent cases.

OPERATIVE TECHNIQUE

At operation a sand bag was placed under the scapula to tilt the patient away from the affected side, thereby rendering the field of operation more accessible. The arm, forearm, and hand were swathed in a sterile sheet to permit of later manipulation. An incision was made parallel to the fibres of the deltoid muscle extending from the edge of the acromion process downward for 2 to 3 inches toward the insertion of the deltoid and crossing the greater tuberosity.

The deltoid muscle was split in the direction of its fibres by combined sharp and blunt dissection. The deeper muscle fibers were carefully pushed aside to expose the roof of the subacromial bursa. The thin wall of this sac was then carefully incised for a distance of 1 inch or more. In only one instance was there any difficulty in entering the sac and that was because of extensive adhesions within the bursa. By retraction of the wound and manipulation, especially rotation, of the arm the bursal walls were separated to permit of an extensive survey of the interior of the subdeltoid portion of the sac. An attempt to illuminate the subacromial portion of the sac by introducing a small electric light in one case was unsuccessful. A curved hæmostat or a finger, aided if necessary by manipulation of the arm, was then introduced to explore the interior of the sac. Eight of the earlier cases were not examined so thoroughly and it is possible some pathological changes may have escaped notice in them but on the whole in the 11 more recent operations the changes involving the bursa itself were relatively insignificant.

In 1 case the bursal sac was completely obliterated by recent fibrinous adhesions which

Fig. 6. Left shoulder. *A* (left). Internal rotation, large shadow. *B*. External rotation. Deposit barely visible in area of acromion shadow.

were readily broken up by the finger. In 3 cases there was some thickening and adhesions of the most dependent portion of the bursa. In 2 of them, a small portion of the sac was excised and in the third, the adhesions were simply separated. In 1 of the above 3 cases and in only 2 others a trifling amount of serous fluid was found. In all but 2 of the cases the calcareous deposit could be seen as a gray patch and usually was palpable to a finger-tip as an area of altered resistance beneath the unopened floor of the bursa.

As the next step in the operation the floor of the bursa was incised over the calcareous deposit. In 4 cases this incision exposed the deposit. Usually, however, a deeper incision had to be made to expose the deposit which was found in the supraspinatus tendon. In 2 cases in which the deposits were situated on the deep aspect of the supraspinatus tendon there was no discoverable superficial index as to their location and they were sought by exploratory incision at the area indicated by the X-ray and by the localized tenderness. In 1 case the deposit was found at the first incision and in the other only after an extensive search.

These deposits varied greatly in consistency. One was not unlike the consistency and color of staphylococcic pus; usually the deposit closely resembled the appearance and consistency of the contents of a sebaceous cyst; in 2 the deposit was rather dry and chalk-like, but not gritty; and in 2 it was granular and gritty.

The major portion of the deposit was lifted out with a blunt curette. Quite commonly the irregular walls of the cavity were smeared or infiltrated with the deposit and had to be trimmed away with knife or scissors. Multiple deposits in some cases called for secondary incisions into the tendon.

The subsequent steps of the operation were carried out in different ways. When there was a definite cavity in the tendon an attempt was made to obliterate it by fine chromic catgut sutures. In a few earlier operative cases the floor and the roof of the bursa were closed with catgut and drainage was not employed. More recently neither the floor nor the roof of the bursa was sutured and a small rubber drainage tube was inserted through the deltoid muscle for 24 hours to provide for oozing blood and serum which often was fairly copious. The

Fig. 7. Right shoulder May 31, 1920. Dense dumb-bell shadow and a larger faint shadow. Skiagram on November 12, 1920, demonstrated complete spontaneous absorption.

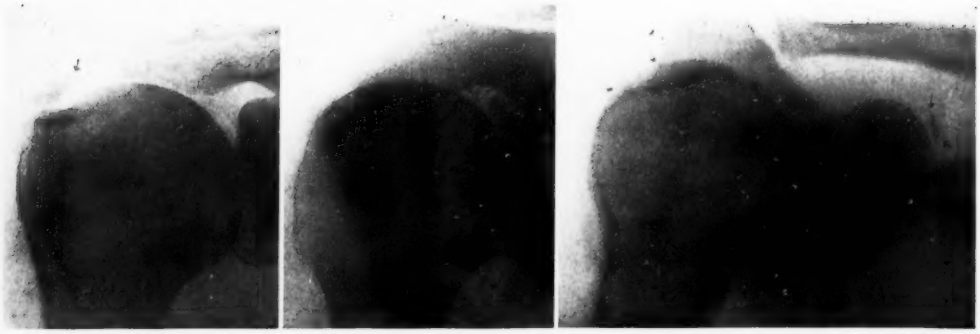


Fig. 8. Right shoulder. *A* (left). July 15, 1922. External rotation. Deposit visible. *B* (middle). Internal rotation. Deposit concealed. *C* (right). October 1, 1924. Almost complete spontaneous absorption. A new deposit has formed at the coracoid process.

deltoid fibers and fascia were united with three interrupted catgut sutures and the skin was closed by a continuous suture. The gauze dressing was held in place by adhesive plaster in preference to a bandage in order to favor early shoulder motion.

The postoperative position of wide abduction, so commonly recommended to prevent adhesions, was found so uncomfortable that it was early abandoned. Practically all these patients had only a bandage sling to the wrist and rested the arm in a position of moderate abduction on a fat, long pillow which rested partly on the mattress and partly across the lower chest and abdomen. They were not restricted to this position but from the first day were encouraged but not obliged to remove the hand from the sling at intervals and extend the elbow; to shift the position of the arm on the pillow; to move the shoulder in any direction they could and even to sit up in bed or in a chair. Beginning at the end of the second or third day they were obliged to begin passive motion in the direction of abduction by interlocking the fingers of the two hands and using the well arm to elevate the bad arm. On the third or fourth day they were required to stand in a doorway or at some offset in the room and gradually "inch" their finger-tips up the wall. This exercise was repeated four or five times daily until they could reach the maximum height level established by the finger-tips of the sound side. During the early days of this exercise the upward climb of the fingers was assisted by using the sound hand to grasp and elevate the affected forearm or elbow and by having the patient step closer to the wall as the fingers ascended.

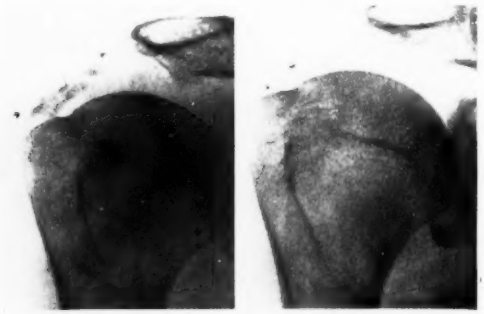


Fig. 9. *A* (left). Left shoulder. Large shadows found at operation to be composed of scores of pin-head-size deposits. Chronic intermittent symptoms for 10 years. Acute exacerbation for 3 weeks. *B*. Symptomless right shoulder. Small deposit.

Beginning on the third day or soon thereafter the patient while sitting rested the arm on a table or on gradually increasing higher piles of books or pillows laid on the table and from time to time depressed the body to increase the range of abduction. Occasionally within one week and frequently within ten or twelve days after operation the full range of shoulder abduction was demonstrated by Codman's maneuver of having the patient stand with knees extended and touch the floor with his finger-tips. About the fifth or sixth days efforts were begun to restore in-

Fig. 10. *A* (left). Left shoulder. External rotation. Deposit visible. *B* (middle). Internal rotation. Deposit concealed. *C* (right). Right shoulder. Small deposits which were shown partially absorbed in 7 weeks and almost completely absorbed in 8 months.

ternal rotation by having the patient pass his sound hand across his back and grasp and make traction on the thumb or wrist of the affected side.

It should be noted that although these exercises are carried out by the patient himself, they are largely of a passive character and do not call for much muscular effort on the part of the affected shoulder. Active exercises are encouraged from the first and insisted upon after the first few days. The exercises were started early to prevent adhesions and were preferably and usually carried out by the patients themselves. Because they could exercise their arms dozens of times daily and could keep their shoulder motions within the range of pain tolerance they made more rapid and more comfortable progress than if they had had to depend upon a nurse or a physiotherapist. For the acute cases in which limitation of motion was due mainly to muscle spasm the preceding exercises were adequate to restore full but somewhat labored motion within 10 days' time.

In all the acute cases relief from the pain immediately followed the operation. Frequently the patients upon recovery from nitrous oxide-oxygen anæsthesia reported within 1 or 2 hours that the old pain was gone and they were usually more comfortable the first night after operation than they had been the few nights preceding it. The patients usually were able to leave the hospital within a week or 10 days. Several physicians left the hospital on the first, second, and third days after operation.

Cases of chronic type with but 2 exceptions were all operated upon during an acute exacerbation. Operation gave them relief from pain only to the extent to which the pain had been increased during the acute exacerbation. The chronic pain they had before the acute exacerbation persisted after the operation and disappeared only after a few to several weeks.

At the time of operation on the chronic cases in which limitation of motion was at least in part due to contracted muscles, only moderate force was used in making shoulder manipulations to restore motion. Moderate force was frequently adequate to restore full motion but sometimes only an incomplete range of motion was obtained. In the chronic cases the patients were instructed to carry out the same postoperative exercises as in the acute cases. In the chronic cases, however, much less rapid progress was made because of the persistence of the original subacute pain. Their own efforts at exercise very commonly had to be supplemented by surgeon, nurse, and physiotherapist both within the hospital,

Fig. 11. *A* (above). Bilateral deposits. *B* (below). Left shoulder in 100 degrees abduction on June 28, 1921. Deposit far under acromion. On July 29, 1924, large left deposit absorbed. Small right and left deposits unchanged.

wherein their average stay was longer, and after going home. Baking, massage and vigorous passive motions had to be employed in many of these cases. A very good device for the patient to use in his home for restoring motion in this and other types of shoulder fixed in adduction consisted of a clothes-line rope and an overhead pulley, each of which was purchased at a ten cent store. At the high points to which the patient could readily reach with his hands two loops were tied in the rope to serve as hand grips. The two ends to the rope were left long to facilitate the grasping of one loop in each hand. A downward pull on the rope by the upward extended sound hand elevated the affected hand and arm and by seesaw movements of the rope the angle of abduction was further increased. Again, this is a form of passive motion which was carried out by the patient himself.

Restriction of outward rotation disappeared spontaneously after operation in the acute cases but required corrective measures in a few chronic cases. I have gained the impression that the pain which persists after operation in the chronic cases clears up less rapidly under prolonged rest than under forced exercises provided the latter are not overdone to the point of aggravating the pain.

In several chronic and a few acute cases massage and special exercises such as swinging Indian clubs and swimming had to be employed to overcome atrophy of the shoulder muscles even after motion was fairly complete.

DISCUSSION OF CASES

I have seen more than 25 shoulders in which calcareous deposits were definitely proved by X-ray examination but in which operation was not performed. Four of these patients had bilateral deposits but had the deposit removed from one side only.

One of these 4 was a physician who had angina pectoris of several months' duration and recurring pain and stiffness of the left shoulder for over 20 years. He had never had any disability in his right shoulder. X-ray showed a definite deposit in each shoulder (Fig. 4). The left shoulder was tender and the right was not. It was difficult for him to differentiate between his angina pain and left shoulder deposit pain. He insisted on operation, which was performed on May 5, 1924, under local anæsthesia. He left the hospital and went to his home at a distance of 200 miles on the third day. Under date of September 25, 1924, he writes that after operation he "had some dull indefinite pain for some time but at this time I am entirely free of any discomfort in that shoulder and have had no discomfort for some time." Concerning the unoperated right shoulder he reports, "I never had any discomfort in my right arm or shoulder until six weeks ago when I was awakened by quite acute pain in the right shoulder which prevented my sleeping until after I had applied heat by means of an electric pad and then I slept through the night. On getting up I had a feeling of soreness and dull aching which persisted for several days, then disappeared, and again made its appearance a week ago, was annoying for three days, and at present seems to have gone. Unquestionably this pain is due to the calcareous deposit in my right shoulder and should it continue I will have deposit removed." An X-ray picture of his right shoulder taken on September 25, 1924, shows the same shadow as pictures taken on March 8, 1924, and on May 4, 1924.

CASE 2. Patient had bilateral deposits proved by X-ray. She had had bilateral symptoms for many years. When she first came under observation she was having an acute exacerbation in one shoulder and the other shoulder was only slightly but rather continuously painful. Motion in both shoulders had been greatly restricted for years. I operated on the acute shoulder and removed the deposit. She was one of my early cases and I gave her an unduly favorable prognosis. With a personal experience based largely on acute cases, I was under the delusion at that time that excision of any deposit was almost synonymous with speedy cure of all symptoms and so advised her. Her acute pain was relieved by operation but the chronic pain persisted and there was not much improvement in shoulder motion during the 2 weeks she was in the hospital. I have not been able to get any news of her condition since she went to her home in a distant town.

CASE 3. Patient was a physician who had several weeks of mild symptoms during which two examinations failed to show any tenderness over the affected left shoulder. He then had a complete remission of symptoms and after taking a bath one night at 10 o'clock he was able to use a bath towel painlessly in the good old-fashioned seesaw way which required full range of shoulder motion in every direction. He then announced to his wife that he was completely free of his trouble. At 3 o'clock of the same night he was awakened by agonizing pain in the left shoulder. His shoulder exhibited the typical hyperacute tenderness and the typical limitations of motion of the acute type. An X-ray picture (Fig. 10A and B) taken the same day demonstrated a deposit in the left shoulder but no picture of the right shoulder was taken. At operation the same day, January 16, 1923, the bursa was found obliterated by fibrinous adhesions which were broken up with a finger and the deposit was excised. He left the hospital on the third day and resumed his work as a skiagrapher on the fifth day after operation. He has had no further trouble in his left shoulder. About 1 year later he began having mild symptoms in the right shoulder and an X-ray picture (Fig. 10C) on January 19, 1924, revealed a definite deposit in the right shoulder. Several pictures taken since then show gradual absorption of the deposit. In the last picture on September 11, 1924, it casts a barely perceptible shadow. The clinical symptoms have been of the milder remittent chronic type and were at about their worst when the last picture was taken.

CASE 4 was a physician who had chronic, fairly constant pain for several months, with marked restriction of abduction of his left shoulder followed by an acute exacerbation and an operation on December 31, 1920. At operation the bursa was entirely free from any suspicion of disease and yet he was one of my worst cases of contracted muscles. By using a considerable amount of force and by snapping "adhesions" during the operation, I was not able to bring his arm out to a full right angle. The deposit was removed. He left the hospital on the fifth day. The acute pain subsided but the original chronic pain persisted for several months. He was an extremely busy out-of-town physician and could not be forced to carry out postoperative exercises but eventually the pain left and the shoulder motion greatly improved. A skiagram was not taken of the opposite shoulder at the time of operation. Recently, I recalled that while he was in the hospital for his operation and subsequently he had complained of a painless stiff right shoulder and for that reason called him on the telephone to request him to have a radiogram taken of the right shoulder. He then stated that for the past few weeks he had been having periodic pains in the previously painless right shoulder, and a skiagram taken on September 27, 1924, showed a pinhead deposit in it.

I have seen 2 non-operative cases of bilateral deposits with unilateral symptoms in the wives of physicians.

One of these patients began 9 years ago to have pain of a fairly constant character and very commonly worse at night in the left shoulder and radiating down the arm. She had marked atrophy of shoulder muscles. A skiagram from one angle only taken in 1917 was negative. A bilateral skiagram (Fig. 11) taken on June 28, 1921, revealed deposits in both shoulders. After this picture was taken the symptoms in the left shoulder began to subside and she has not had any trouble in it for over 2 years. A skiagram taken on September 29, 1924, shows disappearance of the large dense shadow in the left shoulder without much change in the tiny shadows in the right shoulder. She has never had any trouble in the right shoulder.

The second patient slipped coming down stairs, grasped the banister and severely twisted her left arm in early January, 1924. The shoulder was uncomfortable for 1 week and then was free from symptoms for a month when she rapidly developed severe, constant pain causing her to walk the floor night after night. She took "gallons of medicine for neuritis." An X-ray picture of the shoulder taken on July 4, 1924, disclosed a large deposit. Since then her pain has rapidly subsided and she now has only slight pain as the arm approaches the level of the shoulder in full abduction. Her right arm has always seemed perfectly normal but a radiographic examination made on September 30, 1924, revealed a small and hazy but unmistakable deposit in the right shoulder and a complete disappearance of the large dense shadow formerly in her left shoulder.

In one instance I had the unusual experience of discovering accidentally bilateral deposits in a patient who had been skiagraphed for another lesion. Stereoscopic X-ray films were made of the chest of a patient who had metastasis to the lungs secondary to a cancer of the breast. The shadow of a calcareous deposit

was shown in each shoulder but both shoulders were free from symptoms.

Another instance of accidental discovery of deposits occurred recently when a study was being made of the supraspinatus tendon in 6 shoulders in the postmortem room. The fifth shoulder contained four small deposits, two being in the supraspinatus tendon and two in the subscapularis tendon. So far as could be learned the patient had never complained of any shoulder symptoms.

The majority of my patients did not have both shoulders X-rayed; hence other bilateral cases may have escaped detection.

In addition to the preceding 3 cases of bilateral deposits in which there was spontaneous absorption of the deposit in the one shoulder of each case, I have seen 4 other unilateral non-operative cases in which there was a subsequent complete or almost complete disappearance of shadows in from 4 months to 2 years.

One patient with unilateral symptoms of hyperacute form in January, 1919, had a very large shadow (Fig. 5) which had almost completely disappeared in a picture taken 3 months later but he still had painless marked limitation of abduction which soon cleared up following the use of a rope and pulley. Pictures of both shoulders taken on September 28, 1924, were entirely negative.

A physician's wife had a moderately severe acute attack of pain and limitation of shoulder motion with a characteristic point of tenderness. A skiagram (Fig. 7) taken on the third day showed a dense dumb-bell shaped shadow and a larger second definite but hazy shadow. She elected to try rest in bed with arm moderately abducted on a pillow until the acute symptoms subsided. Both shadows had disappeared in 4 months but mild symptoms recurred from time to time during the next 2 years whenever she attempted to play the piano for long periods and playing golf was impossible. She has not had any symptoms for over a year. Her opposite shoulder was never skiagraphed.

A man with mild sumptoms of chronic recurrent type had a small dense shadow which had nearly disappeared a year later, was entirely gone in 18 months and he has not had any symptoms for the past 9 months.

A physician had chronic symptoms with fairly acute exacerbations following an uncertain injury in an automobile accident. He was treated for brachial neuritis for 4 months and then the first X-ray picture was taken and it revealed multiple deposits. Four months later the largest deposit had nearly disappeared, but the pinhead sized ones were unchanged and there was no shadow in opposite shoulder. There was marked improvement in his symptoms but movements such as the overhead swimming stroke cause pain.

Until more convincing evidence is forthcoming, I will continue to believe that the generally accepted view that these deposits can make a miraculous disappearance within a very few days is really due to an X-ray fallacy. I had two cases in which I formerly supposed deposits had disappeared spontaneously within 5 days as proven by X-ray evidence. Films in one angle only were taken in each case. I now suspect that in both instances the skiagram was taken at the favorable angle to show the deposit on the first day and at the opposite angle with the concealment of the deposit by the superimposed bone shadow on the fifth day. Unfortunately I have not been able to trace either the patients or their films in order to check up on this surmise.

One of these 2 patients was a rather curious case in a colored man. He had right shoulder subacute symptoms of sufficient severity to be anxious for operative relief. A single plate of each shoulder proved negative for the painful right shoulder and showed a dense typical deposit in the otherwise normal left shoulder. To eliminate possible error in the labeling of the plates, second pictures were made by another roentgenologist on the fifth day and no traces of the former shadow could be detected in either of the single plates taken of each shoulder. The patient was not operated upon and was not seen subsequently.

In several of my cases and in many reported ones the deposits have apparently made their appearance with amazing rapidity. Absolute proof as to the speed with which these deposits form has not been determined by a first skiagram which is negative and a second which shows a definite shadow. I have had several early cases of shoulder injuries X-rayed hoping to clear up this point but my efforts have been futile.

One of my cases was in a man who developed hyperacute unilateral shoulder pain after driving an automobile on a stormy night. A skiagram (Fig. 1) taken within 12 hours of the onset of symptoms showed a typical large, dense shadow and a corresponding deposit was found at operation 3 hours later.

It has been generally assumed that these deposits do not antedate the onset of symp-

toms, mainly for the reason that it is extremely rare to find them in routine X-ray examinations of shoulders except when the symptoms of the deposits are present. It has been argued that if these fairly common deposits could exist without symptoms they should be encountered frequently in skiagrams taken for other shoulder lesions. Elsewhere in this paper I have shown that (1) symptomless bilateral deposits in the shoulders have been found accidentally in stereoscopic films of pulmonary cancer; (2) a deposit has been present as long as 3 years without causing any symptoms; (3) a deposit has been present but quiescent for 4 months and then caused symptoms; (4) deposits have disappeared spontaneously; and (5) a large dense deposit has been present and has not been demonstrable in an X-ray picture of the shoulder taken from only one angle, especially if it was the common position of having the patient's hand resting on the chest or abdomen. In view of all these facts, I believe that the large dense deposits which are found within the first day or a few days after the onset of symptoms must be instances of pre-existing latent deposits.

The remainder of the non-operative cases were all of the milder subacute type and have drifted away. The nature of their affection was explained to them with suggestions as to treatment and they were advised to return for operation in the event of an acute exacerbation. That so many of them have not returned would seem to imply that many of them experienced the frequent betterment that occurs with the lapse of time.

ETIOLOGY

All recent writers agree that injury to the tendon of the supraspinatus muscle is a definite etiological factor in the production of calcareous deposits, yet they all agree that a history of a definite single trauma is unobtainable in a considerable percentage of cases. According to the commonly accepted theory a partial rupture of the supraspinatus tendon is the injury which is usually responsible for the deposition of the lime salts. Localized tears in this tendon have been observed at operation and postmortem in the absence of deposits and they have also been found at operation at the site of the deposit. In none of my cases was I able to determine that the cavity containing the deposit was due to rupture of tendon fibres. If partial tendon rupture had caused the cavity in any of them all evidence of rupture had been masked by inflammatory changes before operation was performed.

An unusually high percentage of my cases occurred in physicians and physicians' wives. Only a few of this intelligent group were able to assign an adequate trauma as a cause for their symptoms. I believe that in a fairly high percentage of cases the deposition of the lime salts occurs quietly as the result of mild repeated traumata, and precedes, by days, weeks or months, the onset of the clinical symptoms. After the deposit has formed, a very mild trauma may then incite an acute inflammation with a rapid development of the classical clinical symptoms. It also seems probable that some of the supposed partial tendon ruptures found at operation or autopsy may in reality have been tendon defects (cavities) caused by a deposit which may still exist or may have been absorbed. Or a tendon weakened by a deposit may rupture from slight violence in which event the partial rupture is the result and not the cause of the deposit.

As in several of my patients Moschcowitz and others have found microscopically that necrosis of tendon tissue is present in these cases. Moschcowitz has made a careful pathological study of the tissues from Brickner's operative cases and he has found that the lesion is one of "tendonitis," necrosis, and calcification. These findings are borne out by pathological reports in my cases.

The blood supply of tendons is notoriously poor. The tendon of the supraspinatus is so situated that it is frequently subjected to single accidental violent trauma and to milder, many times repeated occupational pinches between the acromion and the head of the humerus in abduction. A partial rupture or a violent contusion either from pinching or from an external blow might directly destroy the scanty blood supply. Milder, frequently repeated traumata of the occupational variety might readily produce the same result in-

directly by chronic inflammation. The localized exæmia would then result in localized necrosis and deposition of lime salts.

The tendon of the supraspinatus is subjected to frequent and repeated traumatism both by muscular action and by direct and indirect violence. The supraspinatus is relatively a very small muscle to be a principal abductor of the shoulder and being attached to the short end of the long widely moving lever represented by the arm is particularly susceptible to lesions resulting from muscular strain. It is therefore not surprising that partial rupture of the tendon may result from sudden unexpected muscular strains, such as occur when the patient suddenly abducts the arm to prevent falling. In many of the reported accidents it is impossible to determine whether the main factor in the injury is the muscular strain or the internal violence due to pinching of the tendon between the humeral tuberosity and the acromion process in the position of abduction. Both forms of violence probably act together in many of these cases and it is possible that the point of partial rupture of the tendon may be determined by the site at which it is compressed between the humerus and acromion. This mechanism would serve to explain why rupture and contusions are so common in the distal end of the tendon and are apparently so rare in the muscle fibers and in the proximal end of the tendon.

Other forms of muscular violence which seem to have played a part in these cases are (1) overuse as in a baseball pitcher; (2) unaccustomed use as in playing baseball without proper training, or in throwing a heavy club into a high fruit tree; and (3) occupational stress with the arms held in slight abduction as in piano players, typewriters and machine operators. In all three of these groups the element of pinching of the tendon cannot be eliminated and it may indeed be the prime factor.

The acromion process might very reasonably be regarded as an essential, although an extracapsular, part of the shoulder joint. Stevens has shown how the short rotators of the arm tend to prevent the upward thrust of the humerus caused by the contraction of the abductor muscles. The normal clearance space between the acromion and the humeral head is very slight and there must be many occasions when the short rotators fail to act perfectly and then the upward thrust of the abducting humerus is arrested by impact against the acromion. Particularly is this impact prone to occur in the arc of abduction between 75 and 85 degrees as the projecting tuberosity passes under the acromion process or coraco-acromial ligament. The acromion process may very reasonably be regarded as an essential part of the shoulder joint itself as it resists the tendency to upward dislocation of the humerus and affords a gliding support to the humeral head during abduction and rotation. In other joints intracapsular articular cartilages cover the bone surfaces which engage in a similar gliding function. The extracapsular subacromial bursa is a poor antifriction substitute for the articular cartilages and undue friction results between the acromion and the tendon covered head of the humerus. The supraspinatus tendon as it winds over the head of the humerus is thus exposed to repeated contusion and friction in a way not encountered with any other muscle or tendon.

Several writers, including Codman, make only passing mention of contusion as the cause of the tendon injury but I believe contusion is a very important factor. Many patients with shoulders otherwise normal both clinically and roentgenographically exhibit marked tenderness over the tuberosities due almost certainly to mild occupational traumata.

Falls on the outstretched hand or on the elbow constitute a form of indirect violence in which the tendon is forcibly compressed between the humerus and the acromion. In these accidents the element of muscular action frequently cannot be eliminated and possibly it may be a contributing or even the main factor in many of them.

The supraspinatus tendon is exposed to direct violence by falls or blows on the point of the shoulder. I think the importance of this mechanism has been overemphasized. Frequently patients get hurt in somewhat complicated accidents and because of the shoulder pain assume they must have been struck in

that region. On cross examination they cannot recall having sustained a blow on the shoulder and the nature of the accidents were such that muscular violence or abduction compression of the tendon or both could have happened. A perusal of case histories does not indicate that this form of direct trauma is at all frequent.

The reported cases of calcareous deposits have not followed severe shoulder injuries. It seems probable that these deposits do occur after severe injuries and are then erroneously regarded as fractures of a tuberosity. I have recently had a dislocation of the shoulder which exhibited an X-ray shadow, which did not permit of differentiation between shell fracture and calcareous deposit. An X-ray picture four months later demonstrated complete disappearance of the shadow. A fracture fragment may have been absorbed but I am inclined to believe that the original shadow was caused by a deposit. If the possibility of a deposit is kept in mind in all shoulder injuries and a careful comparison is made of the tuberosities in stereoscopic pictures of both shoulders, I anticipate that many of the suspected fractures of a tuberosity will turn out to be calcareous deposits. On the other hand, I suspect that many of the cases which have been reported in the past as fractures of the greater tuberosity from muscular violence, often mild in character, were in reality cases of calcareous deposit.

My findings agree with Brickner's views that some factor other than trauma, probably a disturbed metabolism, plays a part in these cases. Brickner points out that "the deposit occurs only in adults; it occasionally is encountered first in one shoulder, then in the other; in some persons the deposit undergoes absorption, in others it persists; although a common affection, many persons using their arms in the same way and subjected to the same influences, never develop it; it occurs among the muscular and athletic as well as the sedentary and asthenic; in females as well as in males; no other hypothesis can explain why in some persons, within a day or two after some mild internal violence or an external injury, the roentgenogram will reveal this characteristic deposition of lime salts above the greater tuberosity of the humerus." I might add that the symptomless development of deposits in the absence of acute trauma is further evidence in favor of metabolic disturbances.

The patients in whom these deposits occur are not of a gouty type. Infection and toxæmia are not factors. Only one of my patients had fever and her temperature rose barely over 100 degrees F. She had an associated œdema of the lower deltoid region and was the only one of my patients that had œdema. She was an early acute case, was 1 of the only 3 of my patients who had an effusion within the subacromial bursa and was 1 of a different group of 3 in whom I found some localized thickening and adhesions of the bursal walls. Cultures from the bursa and from the deposit were negative as were all the cultures taken on several other patients. Many of my cases before the diagnosis had been made were treated for various forms of toxæmia without benefit to the shoulder.

On radiographic examination the calcareous deposits cast a shadow of varying density. The shadow may be thin and hazy or quite as dense as bone. Shadows of both extremes may exist in the same patient. The deposits may be single or multiple, and may be unilateral or bilateral. They vary in size from a pinhead to a silver quarter-dollar. They occupy different positions in relation to the head of the humerus in different patients. They often escape detection when a skiagram is made from only one angle because the deposit shadow is superimposed on the bone shadow of the humerus or the acromion.

I feel greatly indebted to Dr. Henry K. Pancoast, Dr. Donald J. Zulick and Dr. B. P. Widmann for their kindness and courtesy in making the majority of the radiographic examinations in my patients and for testing out various methods of demonstrating the presence of these deposits. Their painstaking examinations often revealed the presence of deposits in patients who had had negative X-ray pictures taken elsewhere within a previous few days. I am convinced that many of these deposits are not shown by the ordinary X-ray examination of shoulders.

In making a radiographic examination of the shoulder for calcareous deposits, Dr. Pan-

coast advises the following procedure: "The rays should be directed from above, downward; and from within, outward in order to show a space between the shadows of the acromion process and the head of the humerus. The calcareous deposit is usually brought still more into prominence by external rotation of the upper arm. From the diagnostic standpoint more information can be gained as to the exact location, by stereoscopic plates made on the Bucky table. This procedure is also more likely to show deposits hidden by the head of the humerus, for the reason that there is more penetration of the bone."

If stereoscopic pictures are not taken, at least two skiagrams should be made in every case. In one the arm should be held in inward rotation to the extent that the hand of the flexed elbow rests on the patient's chest. In the other the arm is rotated externally by flexing the elbow to a right angle and turning the hand as far away from the body as possible which usually means the back of the hand rests on the table on which the patient is lying. In one position or the other the deposit shadow will usually be shown clear of the bone while in the opposite position even dense deposits are frequently obscured or totally hidden by the superimposed bone shadow. Very exceptionally the two shadows may be superimposed in both positions hence it is desirable to make stereoscopic plates before a negative X-ray diagnosis is made. In an unusual case now under observation, X-ray films in both positions of rotation show two small shadows which simulate deposit shadows overlaid by bone shadow. Stereoscopic examination shows that these shadows lying within the humeral head are probably due to localized areas of bone condensation. The patient has the characteristic Codman symptom complex.

Recently in a few cases I have been able to predict which position of rotation will disclose the deposit if one be present by studying the position of the tender area in relation to the humerus and the direction which the X-rays will travel to strike the film. Whether this observation will hold good even in the majority of cases remains to be seen. I believe the tender area below the acromion coincides with the site of the deposit.

The symptom complex in these operative and non-operative cases of calcareous deposit is exactly the same as occurs in other patients in whom the most careful X-ray examinations fail to reveal any evidence of a deposit.

Prior to the appearance of Codman's classical paper on stiff and painful shoulders in 1906, patients with these symptoms were erroneously labeled as cases of brachial neuritis, periarthritis, circumflex nerve paralysis, rheumatism, contusion of shoulder and so on. Similar erroneous diagnoses are very prevalent at the present time. Codman explains these symptoms as being due mainly to an inflammation of the subacromial bursa. In his original paper Codman mentions injuries to the supraspinatus tendon as incidental to injury of the bursa but in his later papers, while still regarding the major lesion as being one of bursitis, he emphasizes the importance of the supraspinatus injury as part of the picture.

Subsequent writers have followed Codman in ascribing the major lesion to the bursa and the minor lesion to injuries of the spinatus tendon. My own limited experience would indicate that the bursal lesion forms the minor feature in these cases and that the injury to the supraspinatus or much more rarely the infraspinatus, tendon constitutes the dominant lesion. The supraspinatus tendon is subjected to practically the same pressure as the bursa between the acromion and the humeral head when the arm passes through the arc of abduction. The tendon is subjected to the same violence as the bursa from direct trauma as from a blow or fall on the shoulder. Indeed by the substitution of the word "tendon" for "bursa" or "bursa and tendon" one can adopt Codman's own description of etiology, causation of symptoms, and treatment and apply it with about equal force to the tendon as being the primary and essential lesion in all the acute and chronic cases irrespective of whether a deposit is present or not. A sore tendon presumably resents being pinched just as vigorously as does a sore bursa. Partial rupture of the tendon is a necessary prelude to a tear in the bursa when the two co-exist from muscular violence. As between these two injuries the rupture would seem to be the more

serious. Although the bursa is fairly adherent to the tendon yet it seems in some cases that partial tendon rupture might occur without tear of the bursa. Particularly is this true in the cases in which a deposit (which presumably occupies the cavity caused by the rupture) is situated on the deep aspect of the tendon and incisions have to be made through intact tendon fibers before the deposit can be exposed. That was the location of the deposit in two of my cases.

In nearly all carefully observed cases the calcareous deposit has been found both by others and myself beneath the bursa, often imbedded within the tendon where it is not in direct contact with the bursa at any point. If these deposits were due primarily to lesions of the bursa it is difficult to understand why many of them are not found in the roof of the bursa. In all of my acute cases removal of the deposit caused immediate cessation of the original pain notwithstanding separate incisions having been made through the roof and the floor of the bursa. If simple tears or contusions of the bursal walls can result in such severe symptoms as these patients suffer, then it would seem logical that the immediate effect of the operation should be an aggravation rather than a cessation of symptoms. Notwithstanding the incisions in the bursal walls not having been sutured these patients soon regained their full range of motion. It therefore seems illogical to assume that either bursal contusion or tear is the likely cause for the months' or years' long duration of symptoms in these patients. The paucity of lesions found in the bursa in my operative cases indicates that bursitis is an infrequent complication rather than a constant co-existent lesion. I think the evidence at the present time is conclusive that calcareous deposits do not originate either in the bursa or in its walls and that the term calcifying subacromial bursitis is a misnomer.

It is very important for surgeons to realize that these deposits are not in the bursa but beneath it. One of my surgical acquaintances was unable to find the deposit in three of twelve operative cases shown by the X-ray because he limited his search to the bursa and did not explore beneath the bursal floor.

The treatment of cases of calcareous deposit will depend upon the stage at which patients apply for treatment. I no longer hold my former radical view that all cases of deposit should be operated upon. Accidentally discovered quiescent deposits do not require operation. If a deposit is causing acute agonizing pain its removal affords the most prompt and most certain method of relief and cure. In between theses two extremes are individual cases of all degrees of severity of pain and disfunction of the shoulder and the decision as to operation is left largely to the patient. The more prolonged or more severe the pain, or the more serious the shoulder crippling, the greater is the need for operation. Awaiting the spontaneous absorption of any given deposit is a very uncertain proposition. Even if it does disappear the symptoms may persist or recur for some months in lessening severity after the deposit has been absorbed. Removal of the deposit, in chronic cases, can be accomplished very comfortably to the patient under local infiltration anæsthesia. Gas-oxygen anæsthesia should be employed for the hyperacute cases in which the slightest touch or movement causes severe distress and for the chronic cases in which it is necessary to manipulate the shoulder to overcome muscular contractures or to break up adhesions.

The chronic cases with contractured muscles that I have operated on usually came during an acute exacerbation brought on by vigorous efforts at passive motion. In the past I have been unduly influenced by the evil effect of passive motion in causing the acute distress before operation and fearing to add to that distress have refrained from making any very forceful manipulations at operation to overcome the restricted shoulder motion. I think this policy has been a mistake as removal of the deposit invariably has relieved the pain of the exacerbation and forcible manipulations after removal of the deposits are not apt to result in any more pain than they would in any other contractured shoulder free from deposit. It therefore seems advisable to use a fair degree of force to loosen up the contractured shoulders at the time of operation.

The use of excessive force in manipulations of the shoulder or of any other joint is not

justifiable. Excessive force means excessive inflammatory reaction and pain, and inability to carry out passive motion over such a long period that the contractures recur. The employment of excessive force in the shoulder may mean fracture of the humerus, dislocation of the shoulder or rupture of the axillary vein. Moderate force by the Sir Robert Jones method of manipulation is frequently sufficient to restore complete range of shoulder motion but if not, the surgeon should be satisfied with partial restoration of motion at the time of operation and later secure full range by having the patient use the overhead pulley and rope. Rarely the patient's efforts will not suffice, in which event it is better to resort to a second and third manipulation under gas anæsthesia with moderate force rather than to use excessive force at the original operation.

Both acute and chronic cases should receive postoperative treatment along the lines previously indicated until there is complete relief from pain, from stiffness of the shoulder, and from muscular atrophy.

I have gained the impression in my nonoperative cases that the distress in acute cases and the acute exacerbation of symptoms in chronic cases are due to an acute inflammation superadded to the chronic inflammation which originally caused the deposit. This acute inflammation with its increased blood supply results in absorption of the deposit irrespective of treatment or lack of treatment. Thus far I have observed absorption of a deposit only after moderately or viciously acute symptoms. Quiescent deposits have neither diminished nor enlarged while under observation. In the cases of calcareous deposits not operated upon, treatment should be directed toward safeguarding the patients from further trauma and keeping them comfortable pending spontaneous absorption. The patients should be instructed to refrain in so far as possible from performing those shoulder motions which their own personal experiences have shown to cause pain. Each time pain is inflicted the underlying lesion is subjected to further trauma with consequent aggravation or prolongation of clinical symptoms.

Intelligent patients soon learn various methods of avoiding pain. For instance an individual with a deposit in the right shoulder will transfer many of his activities to his left hand. The pain caused by putting on a coat or overcoat in the usual way can be avoided by complete insertion of the affected arm into the sleeve first. A woman will place her skirt on hindside foremost so as to fasten its button in front and then rotate the skirt into position.

In those cases in which occupational traumata aggravate symptoms it is desirable, but seldom possible, to have the patients make a temporary change in occupation. The inability of a skilled workman to change his occupation may be a determining factor in favor of operation.

In those patients in whom occupational traumata are due to holding the arms in the abducted position (as in type-writers, piano-players and machine-operators) it will frequently be found that the abduction and traumata can be overcome directly by lowering the level of the machine or by raising the seat, or if the patient stands at work by placing a small platform at the base of the machine. This absolute or relative lowering of the machine level automatically brings the elbows closer to the sides of the chest and thereby removes the abduction element as a cause of the traumata.

Active abduction by increasing the strain on the inflamed supraspinatus tendon increases pain. Passive abduction to an angle of about either 60 or 120 degrees, or rest in abduction at about the same angle, relieves the pain by relieving the strain on the supraspinatus. Abduction whether active, passive, or at rest between 70 and 95 degrees of angulation increases pain by compressing the tendon between the acromion and the tuberosity of the humerus. Rarely a patient learns by his own experience that wide abduction as obtained by tying his wrist to the bed head or resting his hand under his head gives relief at night. Many surgeons endeavor to treat their patients by holding the arm at the 120 degree angle on various forms of splints or plaster casts or by tying the forearm or wrist to the head of the bed. This position shortens the duration of acute symptoms but it is so uncomfortable that I have abandoned it as the patients were prone to consider the treatment

worse than the disease. A 40 to 60 degree angle of abduction can be obtained at night by resting the arm and forearm on a pillow which is placed with one end on the mattress and the other end over the patient's chest and abdomen. The same position is obtained by day while sitting, by resting the arm on a pillow which is placed on the arm of a chair and across the lap or on top of a table. Sedatives may be required to procure sleep at night. If pain is so great as to require morphine, operation should be urged instead.

Physiotherapy in its various forms may be helpful but if used injudiciously may cause marked aggravation of symptoms. Several of my chronic operative cases first came under observation during an acute exacerbation immediately following vigorous massage and passive motion by physiotherapists. Gentle massage combats the tendency to atrophy and may relieve pain but it should not be employed directly over the tender area. Heat in its various forms commonly ameliorates the pain. Many patients find that an electric pad applied to the shoulder at night is a sleep producer. Harris claims to have cured cases of calcareous deposit by diathermia. He apparently refers to symptomatic cures only as he did not follow up his cases by X-ray examinations to determine whether the deposits had been absorbed. I have tried diathermia but am not convinced that it or any other form of treatment has any specific effect in causing absorption of deposits.

The tendency toward the formation of adhesions and the contracture of muscles must be overcome by early and daily resort to full abduction of the shoulder. This can be accomplished painlessly by having the patient stand with knees extended and touch the floor with his finger-tips.

Recurrence of a deposit is a reasonable possibility but thus far I know of no instance in which one has recurred after having been either absorbed or removed by operation.

REFERENCES

Baer, W. S. Bull. Johns Hopkins Hosp., 1907, cxcv, 282.
Brickner, W. M. Am. J. M. Sc., 1915, cxlix, 351.
Idem. J. Am. M. Ass., 1917, lxix, 1237–43.
Codman, E. A. Boston M. & S. J., 1906, cliv, 613.
Ibid., 1908, clix, 533.
Ibid., 1912, clxiv, 708.
Ibid., 1911, clxv, 114.
Ibid., 1912, clxvi, 890.
Dawbarn, R. H. M. Boston M. & S. J., 1906, cliv, 691.
Harris, J. F. J. Am. M. Ass., 1923, lxxxi, 98.
Moschcowitz, E. Proc. N. Y. Path. Soc., 1915, n.s. xv, 1–7.
Painter, C. F. Boston M. & S. J., 1907, clvi, 345.
Stevens, James H. Amer. J. M. Sc., 1909, cxxxviii, 870.

CHRONIC PANCREATITIS AS A CLINICAL ENTITY[1]

By J. WILLIAM HINTON, M.D., New York City
Associate in Surgery, New York Post-Graduate Medical School and Hospital; Adjunct Assistant Visiting Surgeon, Bellevue Hospital

THAT acute exacerbations of upper abdominal pain occurs in a certain per centage of patients having had cholecystectomies for cholecystitis or cholelithiasis is familiar to everyone. The explanation of the recurrent attacks of pain is difficult, and the first impression is a calculus in the common bile duct which was overlooked at the original operation. Quite too frequently these cases are subjected to an exploratory laparotomy during one of the recurrent attacks of pain, only to find the common duct negative for calculi or definite organic obstruction. Judd and Burden (10) report 24 such cases which were subjected to exploratory laparotomy with the pre-operative diagnosis of a calculus in the common duct in 23 cases and acute pancreatitis in one. The exploration failed to reveal a calculus in the common duct and it was also negative for organic obstruction.

It is not uncommon for patients with a chronic cholecystitis to have a history of a sudden severe upper abdominal pain which lasts for a few hours to 24 or 48 hours and then subsides. They may have several of these attacks, which are characteristic of a calculus in the gall bladder or the common duct. When such patients are subjected to a laparotomy, no evidence of a calculus can be detected in either the gall bladder or the duct. The pathological findings are chronic thickened gall bladder with saffron-colored fat and enlarged lymph glands along the cystic and common ducts, with or without induration of the head of the pancreas. The explanation of these recurrent attacks of acute upper abdominal pain seems to result from acute exacerbations of a chronic pancreatitis which has resulted from a pre-existing cholecystitis or appendicitis.

HISTOLOGY

The pancreas is a tubo-alveolar gland which is situated in the upper abdomen, consisting of 3 portions: the head, the body, and the tail. It is sometimes called the abdominal salivary gland. Piersol (13) calls attention to the abundance of the interlobular connective tissue. Hence the compartments of the gland tissue are loosely united and the entire gland lacks the compactness ordinarily seen in large glands. Figure I will show the interlobular connective tissue surrounding a blood vessel of the gland as well as the alveoli.

LYMPHATICS

The lymph drainage to the different portions of the pancreas bears a definite relation to the organs with which the lymphatics are connected. Figures 2 and 3, from the work of Bartels (2), show the lymphatics draining chiefly to the body and tail of the pancreas from the left side of the abdomen. Figure 4, from the work of Franke (8), will show the lymphatics of the gall bladder and biliary tract draining to the head of the pancreas. Figures 5, 6, and 7 also show the lymphatic drainage from the ileocæcal region to the head of the pancreas, and the work of Braithwaite (3) indicates a direct lymphatic drainage from the ileocæcal region to the subpyloric group of lymph glands.

ETIOLOGY OF CHRONIC PANCREATITIS

It would seem from the distribution of the lymphatics that the head of the pancreas is more frequently affected from chronic foci of infection than the remainder of the gland. Sweet (15) states that chronic pancreatitis is the result of inflammation of the interlobular connective tissue of the pancreas, and therefore it is essentially a lymphangitis. Deaver and Pfeiffer (5) report a series of 52 cases of chronic pancreatitis in which they found the head alone affected in 42. In only 9 was the tail involved. In 27 cases there was well marked evidence of involvement of the lymph nodes in the neighborhood of the head of the pancreas. They state that in the majority of cases the entire gland is not involved, but the infection is localized to the triangle of pan-

[1] From the Surgical Service of Dr. Charles Gordon Heyd at New York Post-Graduate Medical School and Hospital.

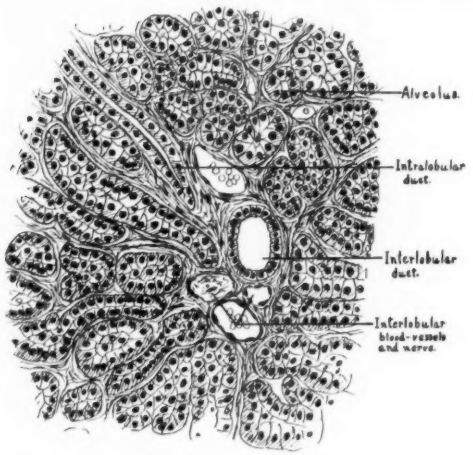


Fig. 1. Section of pancreas showing interlobular tissue with vessels, nerve, and duct and surrounding tubular alveoli.

creatic infection between the duodenum and the converging ducts of Santorini and Wirsung. They attribute the chronic infection to a lymphatic origin. If the infection ascends the pancreatic ducts, the induration should be more general. The lymphatics from the gall bladder drain to Desjardins' triangle of pancreatic infection (6), as has been demonstrated by Franke (idem). Deaver reported 79 patients with chronic pancreatitis at the Lankenau Hospital; 72, 91 per cent, showed evidence of biliary infection, 42, 53 per cent, had calculi, while 30, 38 per cent, showed a non-calculous

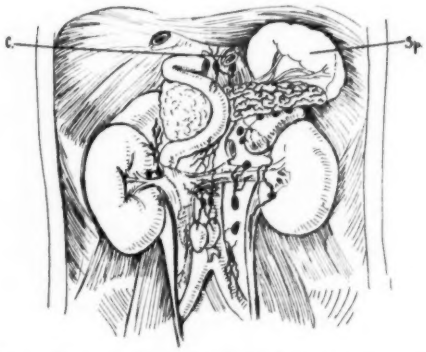


Fig. 3. The relation of the lymphatics of the human pancreas to the regional lymph nodes. *C*, Cardia; *Sp.*, spleen. (From Bartels.)

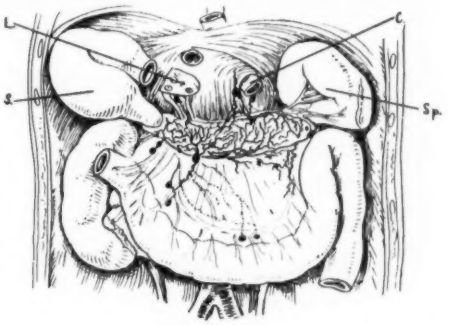


Fig. 2. The relation of the lymphatics of the human pancreas to the regional lymph nodes. *S.*, Stomach turned toward the right; *C.*, cardia; *L*, stump of liver; *Sp.*, spleen.

inflammation. W. J. Mayo (12) reports that 90 per cent of the cases having acute and chronic pancreatitis have been operated upon for infected gall bladders, usually with gall stones. Mann and Giordano (11), working experimentally on goats, doubly ligated the common bile duct and divided it at its entrance into the duodenum. The animals lived from 1 to 30 days. In none was there either macroscopically or microscopically an area with the appearance of acute hæmorrhagic pancreatitis. The results of these experiments emphasized: first, that with the pancreatic duct emptying directly into the common bile

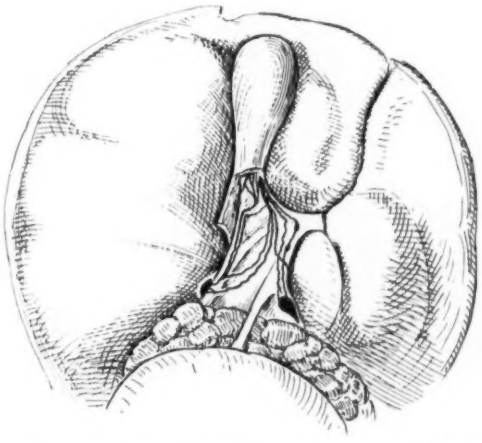


Fig. 4. The relation of the lymphatics of the gall bladder to the head of the pancreas. (From Franke.)

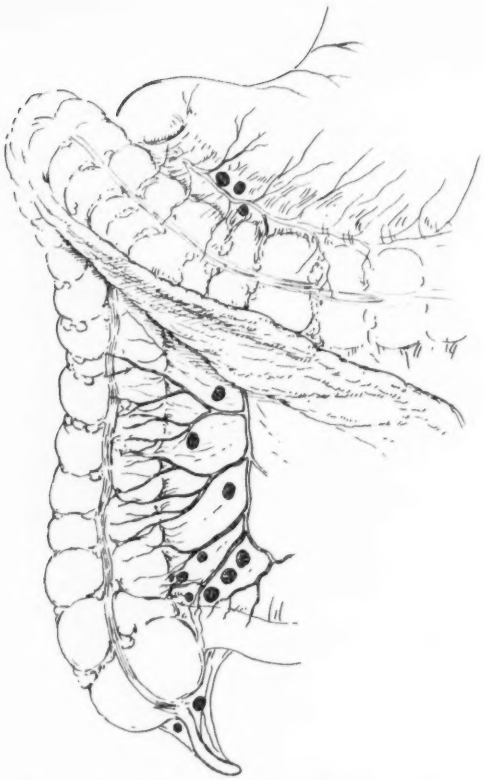


Fig. 5. Showing pyloric congestion and spasm. (From Braithwaite.)

Fig. 6. Diagram showing area of glands injected in the living subject from an ileocæcal gland puncture. (From Braithwaite.)

duct and the latter completely obstructed, bile was not forced into the pancreas except after a considerable length of time; second, that bile did pass into the pancreatic duct and infiltrate the pancreas completely when under the maximum of pressure which the physiological mechanism of the animal could produce. Acute hæmorrhagic pancreatitis was not seen. From the data on the relation of the duct of Wirsung to the common bile duct which is shown in Figure 8, it is anatomically possible for obstruction existing at the ampulla to convert the two ducts into a continuous channel and allow bile to pass in the pancreatic duct in 3.5 cases.

Sweet (16), in working experimentally on dogs, has been able to remove a portion of the pancreas and join the head of the pancreas or the duct to the intestine without danger of pancreatitis, either acute or chronic, for as long as a 11 months' interval. From this, it would not seem that infection ascends by way of the duct.

Eggers (7) gives two possible causes of acute pancreatitis: first, that symptoms are due to infection or ferment action due to the entrance of bile or duodenal contents into the pancreatic duct; second, that it is an infection carried to the pancreas by means of the lymphatics. He leans to the theory that infection as such has nothing or little to do with acute pancreatitis, but that it is due to the action of

Fig. 7. Drawing of an injected postmortem specimen (the needle was placed in an ileocæcal gland). Retrograde flow back to the cæcal wall is clearly shown. (From Braithwaite.)

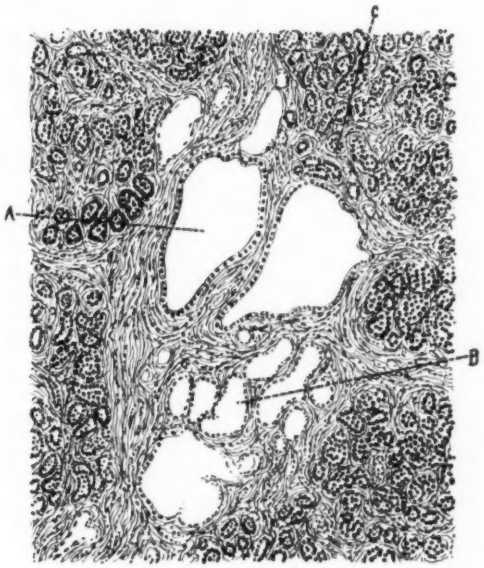


Fig. 8. Section from a fibrosed pancreas showing a combination of centrilobular and perilobular types. There has been obstruction and dilatation of the main ducts, *A*, and their branches, *B*. The lobules are separated by broad perilobular bands of fibrous tissue and the individual acini, *C*, in the lobules are similarly separated by a centrilobular fibrosis. (From Adami and McCrae.)

the liberated pancreatic ferments on the surrounding tissues. In 6 cases reported by him, 5 had gall stones and the sixth had a cholecystitis.

From the above data on the lymphatic drainage of the pancreas, it would seem that the gall bladder and biliary system are the primary factors in producing chronic infection within that area. The appendix and ileocæcal angle play the second rôle, as they have direct connection with the pancreas, as demonstrated from the work of Braithwaite, Craig, and MacCarty (4). The acute symptoms are presumably due to a setting-free of the trypsinogen from the acini cells and the transformation of the trypsinogen into trypsin, through the bacterial action.

PATHOLOGY

Chronic pancreatitis involves the interstitial tissues, and the increased connective tissue is usually confined to the head of the pancreas. From Figure 8 one sees an increase in the fibrous tissues separating the lobules and also the individual ascini of the lobules. Adami and McCrae (1) state that it is to be remembered that in cases of cholelithiasis, we encounter frequently a marked induration, especially of the head of the pancreas, this becoming so firm that the surgeon is apt to mistake it for a new-growth. The clinical and pathological data would indicate that the head of the organ is most frequently affected from chronic infections.

SURGICAL ANATOMY

Due to the location of the pancreas, the anatomical approach to the organ is rather difficult. In surgical conditions of the pancreas, the relation of the organ to the duodenum, the common bile duct, the left kidney and spleen, renders it rather difficult to make a clinical diagnosis of pancreatic involvement. Figure 9 shows the relation of the pancreas to the neighboring organs.

TABLE I.—DATA ON THE RELATION OF THE DUCT OF WIRSUNG TO THE COMMON BILE DUCT

Group	Location of opening of Pancreatic Duct	Number of Specimens	Percentage	Distance from the apex of ampulla of Vater to the divided septum, mm.							
				3	4	5	6	7	8	9	10
1	Separately into duodenum	62	31								
2	2 mm. from the apex of ampulla of Vater	90	45								
3	3 mm. to 10 mm. from the apex of the ampulla of Vater	40	20	28	5	3	0	1	2	0	1
4	Duct absent or reduced to fibrous cord	8	4								
	Total	200									

THE SYMPTOMS OF CHRONIC PANCREATITIS

We are referring to the acute exacerbations of upper abdominal pain that occur in patients following cholecystectomies, and the attacks are more or less similar to those that the patient had previous to operation. These recurrent attacks are usually met with within the first few weeks or months following the cholecystectomy.

PAIN

This is the chief complaint of the patient, and the individual is usually seized with a severe upper abdominal pain which is constant in character. It is difficult for these patients to localize the pain. Occasionally they say it is in the right upper quadrant and radiates to the back. More frequently they state that the pain is on the left side in the region of the left kidney. Due to its location on the left side, it would make one suspect a renal calculus. The intensity of the pain will vary in different individuals, presumably depending upon the severity of the pancreatic involvement. These attacks may last from a few hours to 48 or 72 hours. If the pain is located in the left kidney region at the beginning of the attack, it quite frequently localizes itself in the upper abdomen in the middle of the epigastrium after 48 hours or so. The patients usually feel normal within a few hours after the pain subsides. *Nausea* is not commonly seen in this condition. *Vomiting* is usually encountered, and it comes on approximately within the first hour of the pain, and usually persists during the attack. They may vomit as many as 40 to 50 times within 24 hours. The vomitus is clear or slightly bile tinged and non-offensive. Because of the persistent vomiting and the sudden and severe abdominal pain, one often suspects a high intestinal obstruction, but the general appearance of the patient is not that of one suffering from an intestinal obstruction, and in spite of the severe pain and persistent vomiting, the general appearance does not indicate that the patient is critically ill. *Jaundice* is quite frequently encountered in the severe cases, particularly after they have been incapacitated for 48 to 72 hours. This is presumably due to a compression of the bile duct as a result of the swelling of the head of the pancreas. According to Helly (9), the common duct passes through the head of the pancreas in approximately 62 per cent of cases. Mayo Robson (14) believes that some of the cases of catarrhal jaundice may be due to a mild pancreatitis.

PHYSICAL EXAMINATION

The abdomen is usually not distended and does not have any marked rigidity, but the patient complains of more or less indefinite tenderness over both upper quadrants and frequently in the costovertebral angle, more frequently on the left than on the right side. Eggers was impressed with the lack of physical signs as compared with the severity of the symptoms in his cases. In two of his patients, the house staff ventured to state that they were simply neurotic. The temperature is usually normal, but when elevated it is around 100 degrees F. The blood examination, in the milder cases, will reveal a normal leucocyte count or only a very slight increase in the total count, as well as the polymorphonuclearleucocytes. The urine analysis is negative.

DIAGNOSIS

The diagnosis of this condition can only be made by the process of elimination, and one must exclude renal calculi and bear in mind the probability of a stone in the common duct which was overlooked at the original opera-

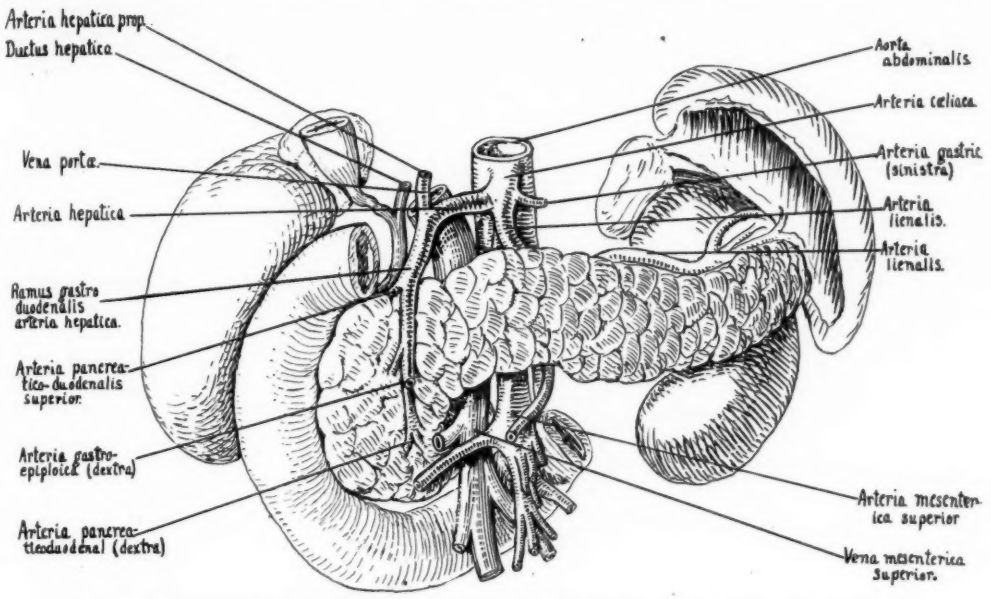


Fig. 9. The pancreas and its relations to the blood vessels and neighboring organs. (From Sobotta.)

tion. In the severer forms of this condition, it is sometimes impossible to make a diagnosis without an exploratory laparotomy. In the milder types of infection, one should suspect the pancreas as the cause of the trouble and advise keeping the patient under observation. In Judd and Burden's series, the findings after an exploratory laparotomy were pancreatitis in 17 out of the 24 cases. The common bile duct was patent in all.

TREATMENT

We should not be too anxious to explore cases that have recurrent symptoms within the first few weeks or months after having had cholecystectomies performed. Some of these cases subside and are permanently cured without further surgical intervention; presumably having removed the primary focus of infection, the pancreatic involvement gradually subsides.

The cases that are explored for calculi in the ducts, and which are negative, continue to have recurrent attacks similar to the ones preceding the laparotomy. Common duct drainage does not seem to have any curative effect on the condition and does not prevent subsequent attacks. W. J. Mayo says it would appear that "as a result of our early postmortem knowledge and tragic experience with an acute pancreatitis, we have been inclined to underestimate the ability of the tissues concerned to localize or cure a large number of acute pancreatic inflammations." This seems to hold true in the recurrent attacks of pain following cholecystectomies which, in a certain percentage of cases, can be attributed to a pancreatic involvement. We should let Nature have its chance to effect a cure.

CONCLUSIONS

1. Some of the recurrent attacks of upper abdominal pain following cholecystectomy seem to be due to acute exacerbation of a chronic pancreatitis.
2. This diagnosis should be arrived at by a very careful process of elimination.
3. Some of these cases spontaneously cure themselves if given the opportunity.
4. Surgery employed in these cases does not seem to cure the condition or prevent subsequent attacks.

BIBLIOGRAPHY

1. Adami and McCrae. Textbook of Pathology. Philadelphia and New York: Lea and Febiger, 1912, p. 595.
2. Bartels. Arch. f. Anat. u. Physiol., Anat. abt., 1904, 299; 1906, 250; 1907, 267.
3. Braithewaite, L. R. The flow of lymph from the ileocæcal angle and its possible bearing on cause of duodenal and gastric ulcer. Brit. J. Surg., 1923, xi.
4. Craig, Winchell, McK., and MacCarty, William C. Involvement of the lymph glands in cancer of the cecum. Ann. Surg., 1923, lxxvii, 698–710.
5. Deaver and Pfeiffer. Ann. Surg., 1913, lviii, 151.
6. Desjardins. These de doct., Par., 1905.
7. Eggers, Carl. Acute pancreatitis. Ann. Surg., 1924, lxxx, 193–209.
8. Franke. Deutsche Ztschr. f. Chir., 1911, Sept.
9. Helly. Arch. f. mikroskop. Anat., 1898, lii, 773.
10. Judd, E. Starr, and Burden, Verne. Non-calculous intermittent biliary obstruction following cholecystectomy. Ann. Surg., 1924, lxxix, 533–537.
11. Mann, Frank C., and Giordano, Alfred S. Arch. Surg., 1923, vi, 1–30.
12. Mayo, W. J. Acute perforations of the abdominal viscera. Surg., Gynec. & Obst., 1919, xxviii, 28–32.
13. Piersol. Normal Histology. Philadelphia: J. B. Lippincott Co.
14. Robson, Mayo. Pancreatic Catarrh and Interstitial Pancreatitis in Their Relation to Catarrhal Jaundice and Also to Glycosuria.
15. Sweet, J. E. The Surgery of the Pancreas. Philadelphia: J. B. Lippincott Co.
16. Ibid., p. 56.

A MANOMETRIC STUDY OF THE CEREBROSPINAL FLUID IN SUSPECTED SPINAL CORD TUMORS[1]

By BYRON STOOKEY, A.M., M.D., F.A.C.S., H. R. MERWARTH, A.B., M.D., and A. M. FRANTZ, A.B., M.D., New York City

From the Department of Neurological Surgery, Neurological Institute, New York

SPINAL cord neoplasms, whether intramedullary, intradural or extradural, sooner or later obstruct the free subarachnoid space and interfere with the circulation of the cerebrospinal fluid. Hence a study of the circulation of this fluid should give data of value in determining the presence or absence of spinal cord neoplasms, by far the commonest form of subarachnoid block.

It has long been known that compression of the veins of the neck, by interference with the venous intracranial outflow causes a rise of intracranial pressure, and, secondarily, cerebrospinal fluid pressure. Also straining, blowing the nose, coughing or deep breathing, etc., increase the cerebrospinal fluid pressure.

When a manometer is attached to a needle in the lumbar sac and the veins of the neck are compressed an instantaneous rise of the fluid in the manometer takes place. Queckenstedt[2] called attention to the fact that when the subarachnoid space is obstructed, the normal rise which follows compression of the veins of the neck does not take place.

In order to determine the clinical value of manometric studies of the cerebrospinal fluid, the present investigation was begun in 1921, on the clinical material of the New York Neurological Institute.[3]

Since then we have studied the manometric readings in more than 50 patients with suspected spinal cord tumors, and in others in whom spinal cord tumor was not suspected. Where spinal cord neoplasm was suspected, the pressure study was made by lumbar puncture alone, for our aim has been to make the lumbar puncture alone yield as much data as possible and to determine the value of such data in the diagnosis of spinal cord neoplasms.

We felt that a thorough manometric study of the cerebrospinal fluid through the lumbar puncture alone had not as yet been made, and that this might enable us to determine the indications for combined lumbar and cistern puncture as advocated by Ayer.[4] When the single puncture does not give sufficient data to permit of an accurate determination being made, we strongly recommend the very excellent procedure of combined cistern and lumbar puncture so skillfully used by Ayer and his co-workers. In this series of cases of suspected spinal cord tumors, the lumbar puncture alone has yielded sufficient data to permit of definite conclusions being drawn in all excepting three. In only these 3 did we find that combined cistern and lumbar puncture was indicated.

We wish to emphasize that manometric studies of the cerebrospinal fluid do not relieve the neurologic surgeon of making a thorough, careful neurologic examination; it is only one part of the neurologic examination. On the other hand, we do not feel that the neurologic examination is complete in any patient in whom a spinal cord tumor is suspected, unless a thorough manometric study of the cerebrospinal fluid has been made.

The mechanism causing an increase in intracranial pressure in compression of the veins of the neck differs from that brought into play by straining, coughing, blowing the nose, or deep breathing, etc. Straining, coughing, and blowing the nose cause a rise in intracranial pressure as well as in intrathoracic and intra-abdominal pressure. The intra-abdominal and intrathoracic pressure causes a rise in the intraspinal pressure by interference with the vertebral and spinal venous circulation, while

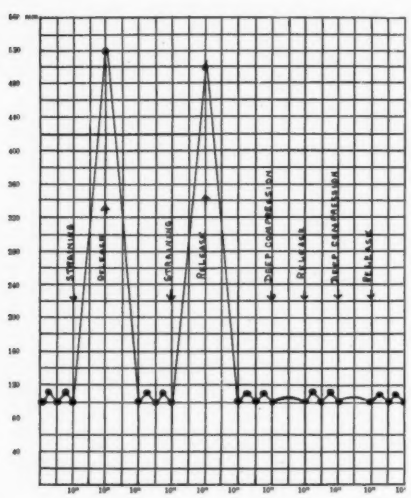
[2] Zur Diagnose der Rueckenmarkskompression. Deutsche Ztschr. f. Nervenh., Leipzig, 1916, lv, 325.

[3] We wish to express our appreciation to the Chiefs of Service of the Neurological Institute, Drs. Elsberg, Kennedy, Tilney, Timme, and Zabriskie, for permission to use their material and for their hearty co-operation, which has made this study possible.

[4] Ayer, J. B. Spinal subarachnoid block as determined by combined cistern and lumbar puncture, with special reference to the early diagnosis of cord tumor. Tr. Am. Neurol. Ass., 1921, 272.

Spinal subarachnoid block. Its significance as a diagnostic sign; analysis of 53 cases. Arch. Neurol. & Psychiat., Chicago, 1923, x, 420.

[1] Read before the Association for Research in Nervous and Mental Diseases, New York, December 29, 1924.



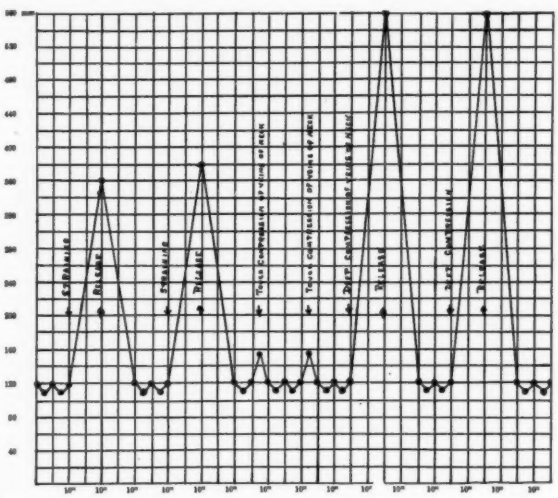


Fig. 1 (left). Manometric chart (E. V. H., No. 27,454) showing complete subarachnoid block. No rise of the cerebrospinal fluid pressure occurred on deep compression of the veins of the neck, whereas a marked rise occurred on straining. This chart shows that there is a dual mechanism involved in the rise of cerebrospinal fluid pressure: (a) Compression of the veins of the neck causing a rise of intracranial pressure; (b) straining causing a rise in intraspinal pressure (see Fig. 13).

Fig. 2. A typical normal manometric reading of the spinal fluid is seen in this chart (No. 27,075) showing normal respiratory and pulse oscillations, prompt rise and fall on touch compression, on straining, and on deep compression.

compression of the veins of the neck interferes with the cerebral venous outflow.

In a normal individual in whom a free subarachnoid space exists, straining, coughing, blowing the nose, etc., cause a rise in the cerebrospinal fluid pressure due to pressure exerted upon the fluid in both the cranial and spinal chambers, while compression of the veins of the neck causes an increase due to an increase in intracranial pressure primarily. This point of difference in the spinal and cranial mechanism of pressure increase is of value in interpreting the manometric readings in cases of spinal cord neoplasm. In some instances where complete subarachnoid block existed we found a marked rise of the cerebrospinal fluid pressure on straining and coughing, but no rise on compression of the veins of the neck. Such a chart is shown in Figure 1, in a patient having a spinal cord tumor verified at operation. On compression of the veins of the neck intracranial pressure was increased and transmitted to the cerebrospinal fluid *above* the tumor. Due to block caused by the tumor this rise in intracranial and spinal fluid pressure *above* caused no rise in pressure in the fluid below the tumor; consequently, no rise in the manometer in communication with the lumbar sac. However, on straining, or coughing, or deep breathing, etc., without compression of the veins of the neck, a marked rise in the fluid in the manometer took place. Since no rise took place on compression of the veins of the neck, which we know increased intracranial pressure above the tumor, and since a rise took place on straining or coughing which increased both intracranial and intraspinal pressure, we may deduce that the rise obtained was due to a rise in intrathoracic and intra-abdominal pressure transmitted to the cerebrospinal fluid below the tumor. Such a result shows well the dual mechanism involved.

Experimentally it was found that when the veins of the neck were ligated in the cat, a rise in cerebrospinal pressure took place; similarly when the vena cava inferior was ligated without ligation of the veins of the neck, a rise also took place, showing that either mechanism is effective in producing a

change in cerebrospinal fluid pressure. However, the rise in the cat is much slower and not as immediate as in the human. This difference may be due to mechanical factors present in the human and not in the cat. Simultaneous compression of the veins of the neck and the vena cava inferior caused a more rapid rise than compression of either of them alone. By simple compression of the veins of the neck in the cat, without exposing them, the cerebrospinal fluid pressure rose, but to a less extent than when abdominal pressure was applied. Abdominal pressure, evenly distributed, was applied to the abdomen by using a blood pressure cuff and a blood pressure apparatus, and caused a greater rise in cerebrospinal fluid pressure than jugular compression.

In our earlier clinical work we tried coughing, blowing the nose, and deep breathing, but found that these were such variable quantities that comparison could not be made. Many of the patients when told to take a deep breath did not seem to know what was meant, and, instead of taking a deep breath, merely threw out their chests and held their breath. Likewise when told to blow their nose, the action and effort expended in the act varied to such an extent that no common factor could be said to exist, and consequently comparisons could not with fairness be made. However, straining, as if at the stool, brought forth in all an effort somewhat more uniform, and while we have had no means of measuring the force exerted, the manometric readings have been sufficiently alike to permit of comparison being made. To increase the spinal fluid pressure through combined intracranial, intrathoracic and intra-abdominal pressure, we have discarded, consequently, the other methods of coughing, blowing the nose, and deep breathing, in favor of straining. The procedures used, therefore, were straining and compression of the veins of the neck.

COMPRESSION OF THE VEINS OF THE NECK

We have found that two types of compression of the veins of the neck may be used, one, firm pressure, sufficient to cause cyanosis of the face, and second, extremely light pressure, which we have called "touch compression."

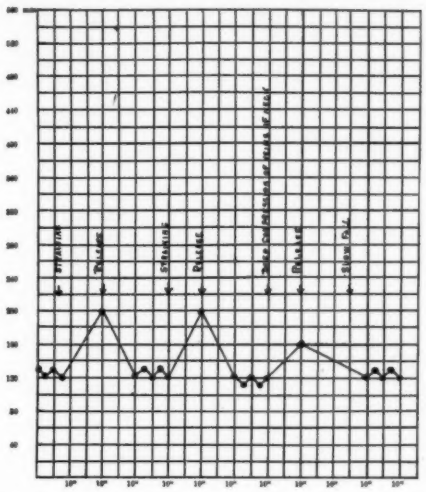


Fig. 3. Manometric chart (P. S., No. 26,393) showing complete subarachnoid block. On deep pressure of the veins of the neck, no rise was obtained, yet on straining a slight rise occurred.

The value of the latter type of pressure we have only lately become aware of and find it of even greater value than the heavier form of compression.

In a normal individual light touch compression will give rise to an immediate fluid wave and cause an appreciable rise in the manometer of from 10 to 30 millimeters, without evoking any straining reaction. The latter causes a rise in intra-abdominal and intrathoracic pressure, and introduces additional factors in the pressure mechanism. Straining or defense reactions may cause a rise of cerebrospinal fluid pressure below the tumor, and unless guarded against, such a rise may be interpreted as the result of compression of the veins of the neck. By touch compression this straining element is not invoked and the results are those of pure compression of the veins.

The extreme delicacy of touch compression makes it a very sensitive test, and we believe that it will prove to be of more practical service than the deeper form of pressure, or in any case, a valuable adjunct to the deep form of pressure.

When firm pressure is applied to the deep veins of the neck and maintained over a period

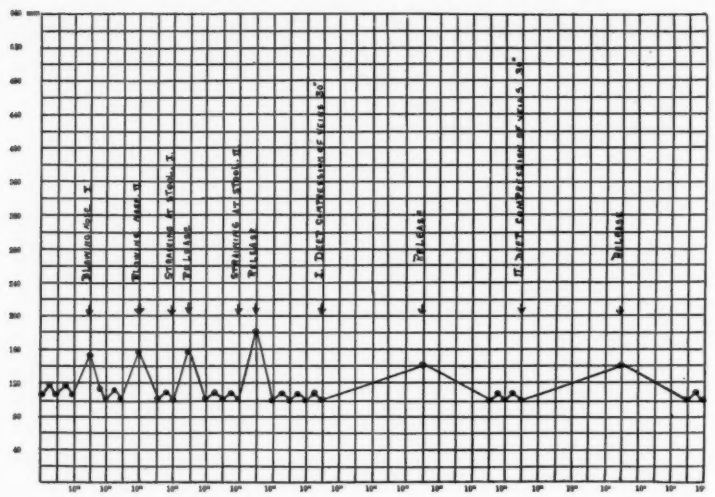


Fig. 4. Manometric chart (C. H., No. 27,572) showing complete subarachnoid block. Very slow and ineffectual rise on compression of the veins of the neck, with a slow return on release of compression.

of from 5 to 10 seconds, a great rise in the cerebrospinal fluid pressure takes place. It is conceivable that this may be sufficient, perhaps, to drive the fluid past a beginning complete obstruction, whereas when light pressure is exerted momentarily the fluid would be less apt to be driven past a block. Consequently touch compression may be considered more delicate.

When deep pressure of the veins of the neck is exerted, co-operation of the patient must be gained and considerable care used to prevent coughing, holding the breath, or straining. Normally, when firm pressure is applied to the veins of the neck an instantaneous and continuous rise of the cerebrospinal fluid in the manometer takes place. Within 10 seconds or less the fluid should rise approximately to 500 millimeters, and as soon as the pressure is removed an instantaneous and continuous fall should take place, such as is seen when the stop-cock of a burette is opened. The fall is generally as prompt as, or more prompt than, the rise. Ten seconds or less may be taken as the normal time for the rise and for the fall. We consider the time element almost as important as the pressure reading.

A typical normal manometric reading of the spinal fluid is seen in Figure 2. The technique which we have now adopted as standard is as follows. The patient is placed on his side in a horizontal position and made as comfortable as possible. After lumbar puncture, a period of 3 to 5 minutes is waited to permit the patient to overcome any pain or fear which may have a tendency to prevent the establishment of the normal level. As soon as complete relaxation is obtained the reading of the cerebrospinal fluid is made. This we have termed the stabilized level. Normal pulse and respiratory oscillations of 1 to 3 millimeters are usually noted.

Each step to be done then is explained to the patient in detail, and he is told to do nothing in anticipation of any command until that command is given—not to move, or strain—to do nothing except remain perfectly quiet until he is told precisely what to do. Unless this is explained the patient frequently anticipates the command and begins to move toward its execution, whereas the execution should be sudden and its duration timed. The patient's co-operation is more readily obtained when he understands in advance what is expected of him.

Touch compression is then exerted over the jugulars. This type of pressure consists of little more than placing the fingers gently over the jugular veins and exerting a moment's pressure. When the fingers are placed over the jugular veins an immediate and instantaneous rise of 10 to 30 millimeters is seen in the manometer. The presence of such an immediate and instantaneous wave generally foretells negative manometric readings in the remaining phases of the manometric tests.

The next step is to have the patient strain as if he were at the stool, straining for 10 seconds. This causes a marked, continuous and steady rise of 200 to 500 millimeters. A wide variation exists in this test due, in the main, we think, to the variation of the force exerted; consequently we consider the facts gained to be of not too great significance, but at times valuable; for example, when a marked rise takes place on straining, with no rise on compression of the veins of the neck, such as has been shown in Figure 1. At one time we thought that, in those cases in which no rise occurred on compression of the veins of the neck, the result of straining might point to a localization of the block, i.e., a small rise might tend to point to a block below approximately the mid-thoracic region, and a considerable rise to a block above this level. This difference on straining was thought to be due possibly to the fact that in a sac shut off below the mid-thoracic level, little fluid and a short column would exist below the block and consequent pressure on the short column would cause little rise; conversely, when pressure was exerted over a longer column with a greater amount of fluid in high placed block, the rise would be greater. While this may perhaps be true theoretically, it was not found to be true in practice in this series of manometric readings.

Our next step is to apply firm pressure over the veins of the neck. As a technical procedure we have found it best to pass the hands around the side of the neck from behind, holding the palms and fingers flat and avoiding the trachea, so as to interfere as little as possible with breathing. With a little practice, pressure can be exerted without causing the patient to strain or cough. By elimination of

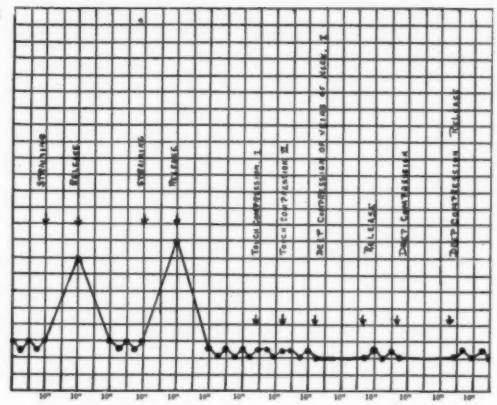


Fig. 5. Manometric chart (S. G., No. 27,759) showing complete subarachnoid block. Essentially the same as Figure 4. The rise on compression of the veins of the neck was minimum, the fall requiring approximately twice as long as the rise.

straining and coughing, a more nearly pure compression of the veins of the neck is effected thus avoiding factors introduced when intrathoracic or intra-abdominal pressure is invoked, which, as has been said, may complicate the picture. Pressure is exerted for 10 seconds. The rise of the fluid in the manometer should be instantaneous and continuous approximately to 500 millimeters or more, and recession likewise immediate and uninterrupted, when the pressure over the veins is removed.

As a rule all steps are done twice to lessen the possibility of error, and in some instances, the second compression of the veins of the neck will bring out manometric changes not revealed in the first.

In our series of cases no *extradural* or *extramedullary intradural* tumor was found in any patient in whom a manometric test, negative in all phases, was recorded. To this rule there was no exception. However, in one patient having an *intramedullary tumor* of the conus a completely negative manometric test was found. In this patient the neurologic examination pointed to a very definite intramedullary tumor of the conus. At operation a very slight symmetrical enlargement of the conus showing a slightly harder consistency was seen and a presumptive diagnosis of intra-

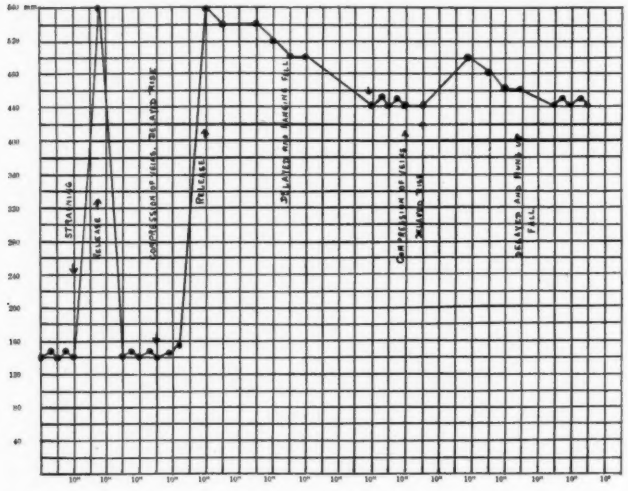


Fig. 6. Manometric chart (No. 27,708) showing incomplete subarachnoid block, Type 1. On deep compression of the veins of the neck a prompt rise was followed by a hanging fall and the establishment of a new level 300 mm. higher than the original level. Second compression of the veins of the neck showed a slight rise from the new level and again a hanging fall. Note the prompt rise and fall on straining, showing increase in rise of pressure *below* the level of the tumor (see Fig. 1).

medullary glioma was made. The rather slight symmetrical enlargement of the cord in this patient did not obstruct the free subarachnoid space. In view of this experience we must, therefore, consider that intramedullary tumors which give rise to symmetrical enlargement of the cord may, in their early stages, present completely negative manometric readings.[1] Such is to be expected from the mechanical factors present in a slight symmetrical enlargement of the cord in this region. Since no type of stasis or obstruction was present with an apparently free circulating fluid, we feel that examination of the fluid at different loci would not have revealed any noteworthy difference in the fluids from different levels, particularly since the lumbar fluid did not show any globulin and only five cells.

MANOMETRIC TESTS INDICATING COMPLETE BLOCK

In all of the patients in whom manometric findings indicated complete block, definite obstruction of the subarachnoid space was found in those operated upon (Fig. 1). In this group of 14 patients showing positive manometric findings, two refused operation and consequently are unverified. However, the neurological examination left little doubt as to the diagnosis. In the remaining 12 operated upon, neoplasms were found in 11, and in the twelfth an extradural tubercular cyst was found blocking the subarachnoid space and compressing the spinal cord quite the same as if it had been an extradural spinal neoplasm. Thus in all cases of this group verified by operation, obstruction of the subarachnoid space was confirmed.

This evidence of obstruction of the free circulation of cerebrospinal fluid was, however, further substantiated by the fact that, in each instance, a marked globulin increase was noted. Xanthochromia was present in five. Xanthochromia without a marked increase in the globulin content was not seen, but a marked globulin increase without xanthochromia was common. In view of the opinion held by some that xanthochromia is not found in intramedullary tumors, it is inter-

[1] Since this was written a second patient showing no intramedullary tumor of the mid-thoracic region presented an almost negative manometric reading, so nearly negative that it was called such, and the conclusion reached, so far as the manometric test showed, that no block existed, yet at operation an intramedullary tumor was encountered.

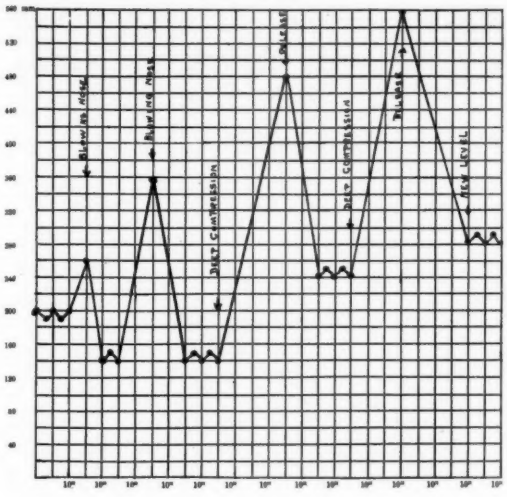


Fig. 7 (left). Manometric chart (No. 26,431) showing incomplete subarachnoid block, Type 1. Prompt rise on compression of the veins, followed by prompt fall, with the establishment of a new level 100 millimeters higher than the original. On second compression of the veins of the neck a prompt rise and a slower fall took place, followed by the establishment of another level, 40 millimeters higher.

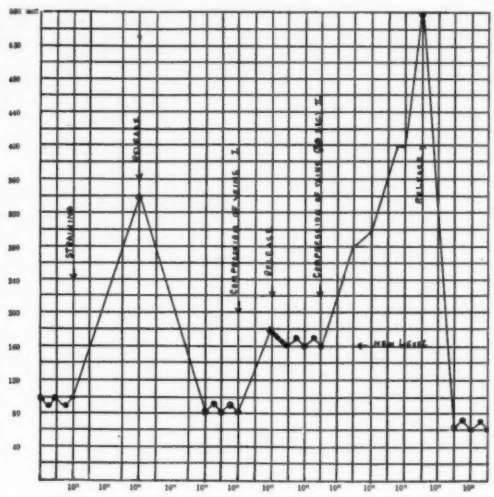


Fig. 8. Manometric chart (No. 27,642) showing incomplete subarachnoid block, Type 2. On compression of the veins of the neck a rise of approximately 100 millimeters took place, with the establishment of a new level 100 millimeters higher than the original level. On second compression of the veins of the neck an irregular and labored rise requiring 30 seconds in place of 10 seconds occurred, followed by a prompt fall approximately to the original level.

esting to note that in both intramedullary tumors in this series xanthochromia was present.

In reviewing the neurologic examination of the patients in whom complete subarachnoid block was found, we feel that the diagnosis could have been made from the neurologic examination alone by any one with experience in the clinical course of spinal cord neoplasms, irrespective of the manometric findings. The manometric findings merely offered confirmatory evidence in support of the diagnosis. Since lumbar puncture must, in any event be done, it is of course reassuring to have further evidence in support of the diagnosis, especially when such evidence can be so readily obtained. We feel that when *positive* manometric findings are obtained through lumbar puncture, no further information of additional value can be gained from combined cistern and lumbar puncture. A typical positive manometric chart is shown in Figure 3. We have found that the positive charts indicating complete block follow approximately two types, namely: one in which compression of the veins of the neck causes no rise at all, or essentially no rise, and second, one in which the rise on compression of the veins of the neck is minimum—seldom more than 50 to 60 millimeters. It has been difficult to determine whether or not the minimum rise in this latter group is really due to compression of the veins of the neck or to straining or holding the breath, which in some patients is apparently an unavoidable association. Practically this slight rise is of little significance, since such manometric reading can, in no wise, be confused with the decidedly marked rise found when no obstruction exists. Occasionally the slight rise associated with compression of the veins of the neck is sustained a minute or more, recession being extremely slow and irregular, at times not to the old level but a new one, 10 to 20 millimeters higher.

The pressure readings in the group of complete subarachnoid block are shown in Table I. The average initial pressure in the group of complete subarachnoid block was found to be approximately 90 millimeters and the average pressure on straining 210 millimeters—a

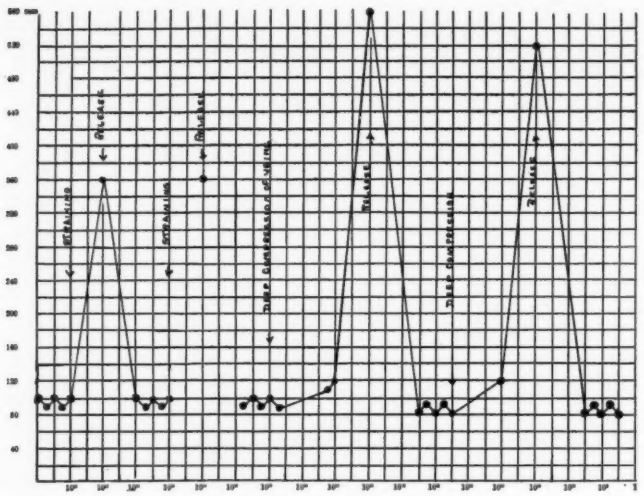


Fig. 9. Manometric chart (No. 27,491) showing incomplete subarachnoid block, Type 3. On deep compression of the veins of the neck a delay in the rise occurred. The fluid hesitated an appreciable time and then promptly rose and fell.

TABLE I.—MANOMETRIC FINDINGS INDICATING COMPLETE SUBARACHNOID BLOCK

A. Complete Block; Verified

Name	Level of fluid in manometer in mm.	Rise on straining, blowing nose or deep breathing	Rise on compression of veins of neck	Fluid Glob. Color	Level	Pathology
R. Y., 27017	170 mm.	320 mm.	80 mm.	4+ Yellow	C III–V	Glioma intramedullary
P. S., 26398	140	180	50	4+ Yellow	C VI–VIII	Glioma intramedullary
A. D., 25183	100		0	3+ Clear	C I–II	Endothelioma
C. H., 27572	110	160	40	3+ Yellow	C IV–VI	Neurofibromatosis
L. G., 27759	70	160	0	4+ Yellow	Th I–II	Sarcoma extradural
M. S., 27896	160		0	3+ Clear	Th II–V	Sarcoma extradural
J. F., 26932	0	100	0	3+ Clear	Th IV–V	Sarcoma extradural
M. D., 26590	40	80	20	4+ Clear	Th V	T. B. extradural
E. P., 26480	100	190	20	4+ Yellow	Th V–IX	Sarcoma extradural
E. V. H., 27454	100	500	0	2+ Clear	Th VII–IX	Sarcoma
F. Z., 27921	90	180	0	2+ Clear	Th IX	Endothelioma
M. D., 26146	140	200	30			Endothelioma
Average	110 mm.	207 mm.	40 mm.			

B. Complete Block; Unverified

M. A., 27265	100 mm.	190 mm.	0 mm.	2+ Clear	C VIII	
A. P.	80	300	40	1+ Yellow	Th V–VI	
Average	90 mm.	245 mm.	40 mm.			

In this group combined cistern and lumbar puncture is not indicated.

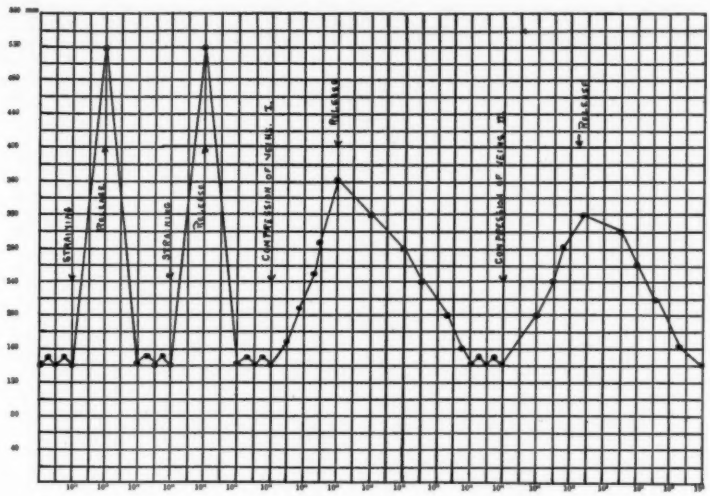


Fig. 10. Manometric chart (No. 26,954) showing incomplete subarachnoid block, Type 4. On deep compression of the veins of the neck a slow and labored rise and fall occurred. Type 5 is similar to Type 4, the rise and fall being smooth and continuous, but requiring a longer time than normal for both the rise and for the fall.

TABLE II.—MANOMETRIC FINDINGS INDICATING INCOMPLETE SUBARACHNOID BLOCK

A. Incomplete Block; Verified

Name	Level of fluid in manometer in mm.	Rise on straining, blowing nose or deep breathing	Rise on compression of veins of neck	Type of rise	Cerebrospinal fluid	
					Glob.	Color
O. S., 27491	100 mm.	380 mm.	540 mm.	II		
C. D., 26954	140	510	380	V	o	Clear
K. D., 27546	160	260	500	I	2+	Clear
F., 26431	160	370	480	I		Clear
A. H., 27731	100	350	470	IV	o	Clear
E. L., 27699	110	460	510	I	2+	Clear
J. C., 25249	100		220	I	o	Clear
Average	124 mm.	388 mm.	442 mm.			

B. Incomplete Block; Unverified*

J. S., 27642	100 mm.	240 mm.	500 mm.	I	o	Clear
G., 27200	230		300	I	o	Clear
H. G., 27641	100	340	440	II	o	Clear
S. S., 27358	160	510	510	I	4+	Yellow
Average	147 mm.	363 mm.	437 mm.			

*Operation refused by patient or postponed.

In all of this group showing incomplete block of different types the free subarachnoid space was definitely compromised in all the verified cases. In the remaining cases sufficient signs were present to make us feel that definite pathology was present. In other words, incomplete block was not found in any case in which definite signs did not exist.

TABLE III.—MANOMETRIC FINDINGS INDICATING INCOMPLETE SUBARACHNOID BLOCK

I. Diagnosis Certain without Manometric Tests; Manometric Tests Confirmative*

		Level	Pathology
S. S., 27358	Unverified	Th X	
O. S., 27491	Verified	C VII	Sarcoma?
E. L., 27699	Verified (X-ray)	Th VII	T. B. spine
F., 26431	Verified	Th II	Extradual sarcoma
J. D., 25304	Verified	Th VII–VIII	Meningomyelitis
			Adherent arachnoid

*In this group combined cistern and lumbar puncture is not indicated.

II. Diagnosis Doubtful. Diagnosis Established by Manometric Tests*

C. D., 26954	Verified	C VII	Glioma, intramedullary
K. D., 27546	Verified	Th IV–V	Endothelioma
G., 27200	Unverified	C VII	Malignancy (secondary)
J. O'N., 25249	Verified	Th VI–VIII	Meningomyelitis
			Adherent arachnoid

*In this group combined cistern and lumbar puncture is not indicated.

III. Diagnosis Doubtful; Manometric Tests Suggestive*

A. R., 26186	Unverified		
H. G., 27641	Unverified	Th V	
A. H., 27731	Verified	Conus	Glioma intramedullary

*In this group combined cistern and lumbar puncture is indicated.

difference of 120 millimeters over the initial pressure, while the rise on compression of the veins of the neck was only 20 millimeters. This is in marked contrast with the manometric readings in normal individuals or those with incomplete block. The pressures of those showing incomplete block is seen in Table II. The average initial pressure in this group was found to be approximately 120 millimeters, with the average pressure on straining 330 millimeters, or a difference of 210 millimeters over the initial pressure, while the rise on compression of the veins of the neck was 440 millimeters. Thus the pressure readings in the latter group approximate those found in normal individuals in whom the arachnoid space is entirely free. However, the *time* required for the rise, and the *manner* of the rise in those showing incomplete block is sufficiently different from the normal so as not to be confused with them.

The pressure in the normal group varies from 100 to 250 millimeters, with pressure on straining of approximately 300 to 500 millimeters and on deep compression of the veins of the neck of 400 to 550 millimeters. In a normal individual the rise and the fall are instantaneous, continuous and smooth, without interruptions or the establishment of a new level.

MANOMETRIC TESTS INDICATING INCOMPLETE BLOCK

The manometric findings in this group stand intermediary between the completely positive and the completely negative groups and represent, pathologically, spinal cord neoplasms or other forms of subarachnoid block in less advanced stages where the subarachnoid space is not completely shut off. Stasis of the cerebrospinal fluid and marked interference with the spinal venous circulation has not yet taken place. From a diagnostic standpoint, this group includes those interesting and more difficult cases in which the neurologic examination alone is apt to leave the diagnosis in doubt. It is in this group that manometric tests may be a very

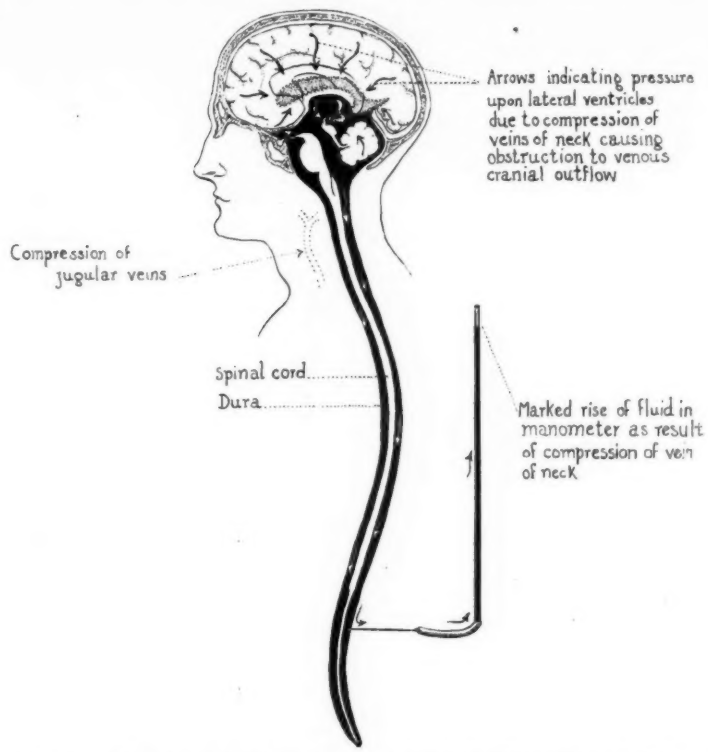

Arrows indicating pressure upon lateral ventricles due to compression of veins of neck causing obstruction to venous cranial outflow
Compression of jugular veins
Spinal cord
Dura
Marked rise of fluid in manometer as result of compression of vein of neck

Fig. 11. Schematic drawing showing mechanism of alterations in the cerebrospinal fluid pressure. A marked rise in intracranial pressure takes place when the venous cranial outflow is obstructed by compression of the veins of the neck. The cerebrospinal fluid is forced out of the cranial cavity into the spinal subarachnoid space, causing a marked rise in the manometer in connection with the lumbar sac.

great help in determining the presence or absence of incomplete block, and in influencing the conclusion for or against exploratory laminectomy.

In reviewing the neurologic examinations alone, we found that of the 13 cases included in this group in 6 the diagnosis of subarachnoid block seemed warranted without manometric tests, the latter tests in the main serving as confirmatory evidence in support of the presumptive diagnosis. From this standpoint such evidence is, of course, reassuring as an additional factor indicating the advisability of an exploratory laminectomy.

In four of the group showing incomplete block the diagnosis was established by the manometric examination; the neurologic examination having failed to give sufficient evidence to warrant the presumptive diagnosis of subarachnoid block. One of these patients showed essentially a cervical root syndrome without any definite signs of cord involvement. Had it not been for the manometric examination we feel that the operation on this patient might have been postponed until more advanced signs had manifested themselves. However, in view of the manometric examination, a more positive stand could be taken and operation was performed. A very large fusiform enlargement of the cord, presumably an intramedullary tumor, was found. A needle inserted into the cord showed yellow fluid which coagulated almost immediately.

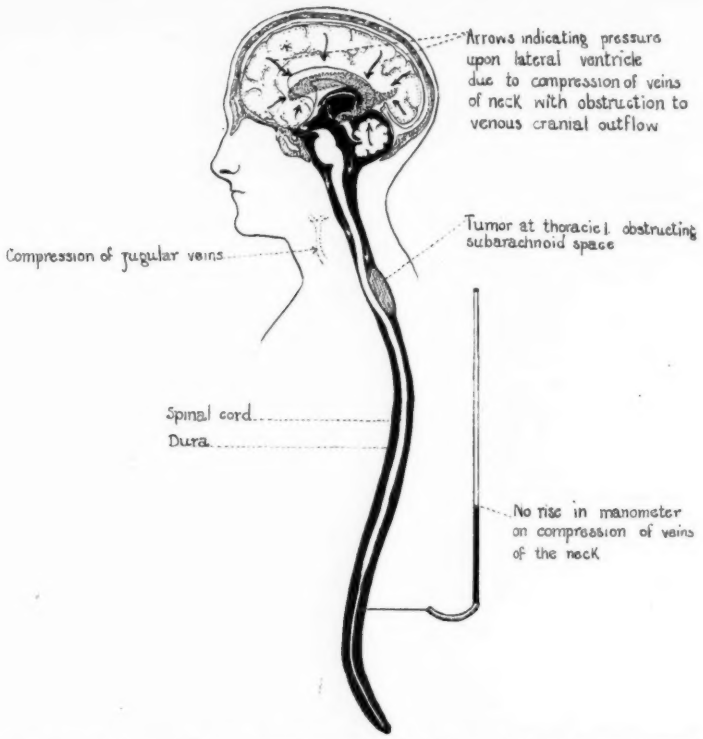

Arrows indicating pressure upon lateral ventricle due to compression of veins of neck with obstruction to venous cranial outflow
Tumor at thoracic I obstructing subarachnoid space
Compression of jugular veins
Spinal cord
Dura
No rise in manometer on compression of veins of the neck

Fig. 12. Schematic drawing showing mechanism of alterations in the cerebrospinal fluid pressure in the presence of a subarachnoid block. Compression of the veins of the neck forces fluid into the spinal subarachnoid space; however, a block in the subarachnoid space does not permit transmission of the increased pressure of the fluid *above* the tumor to the fluid *below* the tumor; consequently, no rise in the manometer in connection with the lumbar sac takes place.

Without the manometric tests we feel that most of the patients in this group may not have been explored until a later time, when perhaps the chances for complete return of function would have been materially lessened. We recall vividly the history of a patient now in the Neurological Institute in whom a spinal cord tumor, readily removable, was taken out 2 years after his first presentation to the staff conference. While spinal cord tumor was considered at the first presentation 2 years previously, sufficient neurologic evidence was not available to warrant an exploratory laminectomy. No manometric test had been done, and to this extent the neurologic examination was incomplete. Had manometric readings been taken we feel that evidence would have been gained which would have warranted an exploration in spite of the incomplete neurologic findings. This patient was lost track of for 2 years, and on return for examination marked evidence of spinal cord tumor was present; but so great a destruction of the cord had occurred that in spite of removal, no return of function has taken place.

In an additional series of three of this group showing incomplete block, both the neurologic and manometric examination left the diagnosis still in doubt. While the manometric changes were suggestive of incomplete block, the evidence was so slight that the diagnosis of incomplete block was considered too doubtful to warrant an exploratory laminectomy.

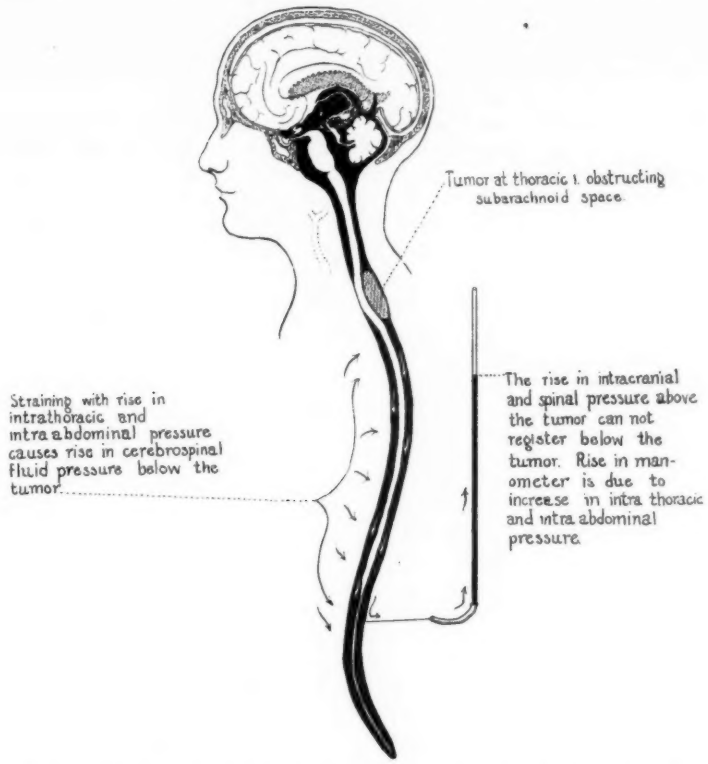

Tumor at thoracic 1. obstructing subarachnoid space.
Straining with rise in intrathoracic and intra abdominal pressure causes rise in cerebrospinal fluid pressure below the tumor.
The rise in intracranial and spinal pressure above the tumor can not register below the tumor. Rise in manometer is due to increase in intra thoracic and intra abdominal pressure.

Fig. 13. Schematic drawing showing mechanism of alterations in the cerebrospinal fluid pressure in the presence of a subarachnoid block. Straining causes a marked rise in intracranial, intrathoracic, and intra-abdominal pressure; consequently, in a free subarachnoid space all these factors are involved to produce a rise in the cerebrospinal fluid pressure on straining. However, when a subarachnoid block is present the rise due to increased intracranial pressure is ineffectual, since the fluid *above* the tumor is shut off from the fluid *below* the tumor. The rise in the manometer on straining is therefore due to the intrathoracic and intra-abdominal pressure exerted on the cerebrospinal fluid *below* the tumor.

When the manometric readings are only slightly suggestive and the neurologic examination is also indefinite, we feel that combined cistern and lumbar puncture is definitely indicated. Of the 50 cases of suspected spinal cord neoplasms included in this study, we have felt that combined puncture was indicated in only these three. Our opinion is that combined puncture should be reserved for those cases in which both the neurologic examination and manometric readings are indefinite. Combined lumbar and cistern puncture certainly is not indicated in those cases in which frankly negative manometric tests in all phases are found. Combined puncture certainly is not indicated in those cases in which a frankly positive manometric test is found. Nor is combined puncture indicated in the other intermediate group in which definite evidence of incomplete block is gained by the manometric examination. But on the other hand, combined puncture is definitely indicated in the remaining very small group in which neither the neurologic nor the manometric examination frankly results in neither completely negative findings

nor those indicating complete or incomplete block. In this *subgroup* of those thus suspected of having *incomplete* block, we feel that the greatest help is to be gained by a combined cistern and lumbar puncture, as advocated by Ayer and his co-workers. Manometric charts indicating incomplete block are shown in Figures 6, 7, 8.

We have attempted to group the various incomplete charts into five types, but the distinction between each can not be too sharply drawn. The first three types, and variations of them, we feel are indicative of incomplete block, but types 4 and 5 are distinctly less suggestive. In the latter two types the diagnosis of incomplete block should therefore be confirmed by combined puncture before recommending exploratory laminectomy.

CONCLUSIONS

1. In suspected spinal cord neoplasms manometric readings of the cerebrospinal fluid through lumbar puncture should be a routine procedure. Examination is incomplete without a manometric study.

2. Manometric studies of the cerebrospinal fluid through lumbar puncture (without cistern puncture) may indicate complete subarachnoid block, incomplete subarachnoid block, or the presence of a free, unobstructed subarachnoid space.

3. In all patients operated upon in whom the manometric tests indicated *complete* subarachnoid block, some form of spinal cord neoplasm was found.

4. In all patients in whom the manometric tests indicated *incomplete* subarachnoid block, either a spinal cord neoplasm or some other form of subarachnoid block was found.

5. Negative manometric readings were found in two patients having an early symmetrical enlargement of the cord, presumably an intramedullary tumor, which, however, did not interfere with the free circulation of the cerebrospinal fluid. In all others exposed the manometric findings were substantiated at operation.

6. Combined lumbar and cistern puncture is indicated when manometric studies through the lumbar puncture alone do not permit of definite conclusions being drawn. In our experience combined lumbar and cistern puncture was indicated in only 3 cases out of 50 suspected spinal cord tumors.

RADIATION THERAPY IN DEEP SEATED MALIGNANT DISEASE[1]

By G. E. PFAHLER, M.D., Philadelphia

RADIATION therapy in malignant disease was at first used only on the recurrent and the hopelessly inoperable cases. In some of these, striking effects were obtained. Gradually more and more of the primary, superficial cases were referred for treatment, until now radiation is the method of choice in the treatment of the superficial and non-infiltrating carcinoma.

Likewise, in deep seated malignant disease, only the recurrent, metastatic, or hopelessly inoperable, cases were originally referred for treatment. Many of these have shown striking results, and some have shown permanent recovery. This has established confidence and gradually the entire profession is recognizing the value of radiation. More and more primary deep seated malignant disease is being subjected to radiation early.

The recognition of radiotherapy is national and international, as is indicated by the fact that no hospital is today considered fully equipped unless the radiological department is prepared for both superficial and deep radiotherapy. The American College of Surgeons, in its last report[2] states: "Superficial and deep therapy is advisable when possible and practical. Supervision through a medical roentgenologist is essential." Ewing very properly says: "The rapid adoption of radiotherapy must stand as evidence of the intellectual honesty of the medical profession. Yet there is still an undercurrent of antagonism which reaches the public with much force, greatly impedes progress, interferes with the spread of knowledge, retards the acquisition of equipment and prevents many from receiving the benefits now available."

The fact that so much progress has been made in the short period of 25 years indicates that the value is inherent in the radiation. While most of the advance work has been done by men who have literally devoted their lives and entire energies to the subject, still much of the work has been done by wholly untrained and inexperienced men. Naturally, as a result, you have all seen the effects of much poor and unscientific work. Your local radiological colleagues, with their earnest and scientific zeal in radiotherapy, have convinced most of you of its value, or I would not have been invited to present this subject before you.

In superficial lesions, the problem is relatively simple. In deep lesions, however, it is much more difficult for one must always aim to preserve the normal tissues and the function of essential organs through which the radiation passes. Otherwise the problem would be merely a physical one, to be solved chiefly by the physicist. In fact, it has been the impression among many, within recent years, during which time one has heard much of the so called "deep therapy," that a physicist, or a few hours instruction by a physicist, was the most essential requirement.

A good radiotherapist must have a knowledge of general medicine (the more the better), and of general pathology and special pathology. He must be well informed in physics, electricity, and mechanics, and he must have an imagination that will enable him to picture in his mind the anatomy, the distribution of the disease and the distribution of the rays, as each beam is directed into the body, so that he can make the rays produce the greatest possible effect on the disease and the least upon normal tissue and organs. Therefore, the greater the knowledge and skill of the radiologist, the better will be the results.

SUSCEPTIBILITY OF TUMORS

It has long been recognized that tumors vary to a considerable degree in their susceptibility to radiation. Even tumors of the same type vary considerably. Ewing says: "In general, tumors derived from embryonal cells and retaining embryonal characters, even when growing rapidly, are, as a rule, particularly susceptible to radiation, and in this

[2] Surg., Gynec. & Obst., 1924, November.

[1] Presented by invitation before the Toledo Academy of Medicine, December 5, 1924.

group some of the most remarkable and paradoxical of the radium cures have been recorded." Most of you, and all of us radiologists, have seen patients with most extensive disease—apparently hopeless cases—respond beautifully and go on to recovery, while others with comparatively little disease show no response.

Ewing (2) classifies tumors according to their radiosensibility, as follows:

1. Lymphoma: lymphocytoma, lymphosarcoma, myeloma.
2. Embryonal tumors: carcinoma of the testes and ovary; basal cell carcinoma.
3. Cellular anaplastic adult tumors: "round-cell" carcinoma, diffuse carcinoma.
4. Desmoplastic tumors: carcinoma simplex, fibrocarcinoma, squamous carcinoma.
5. Adenocarcinoma: adenoma of the uterus, intestine, breast, etc.
6. Fibroblastic carcinoma: osteosarcoma, neurosarcoma.

The microscopical changes in the cells under the influence of radiation consist of swelling, hyperchromatism, vacuolar degeneration and solution or fragmentation of nuclei, hydropic swelling, vacuolation and solution of the cytoplasm. Mitotic nuclei are particularly vulnerable, the chromosomes splitting, the spindle threads disintegrating, and the whole cell undergoing relatively speedy solution. The surrounding stroma exhibits hyperæmia, slight serous exudation, outwandering of leucocytes, and growth of new capillaries, which in many instances probably plays a prominent part in the removal of tumor cells."

Radium is probably more selective in its action on tumor cells than the X-rays. By his experiments on the larva of frogs, Friedrich has found that the radiation from radium in like quantity has about three times the biological effect as compared with that from the roentgen-rays. My clinical experience will confirm this observation. Therefore, when one can choose, it is advisable to use radium for the destruction of malignant disease whenever it can be brought in direct contact with the disease. The X-rays, however, are, preferable when the radiation must be carried to some depth, through other tissues in order to reach the tumor, or when the disease must be destroyed at a depth of more than 3 centimeters, (1) because the direction of the radiation from radium is most difficult to control, while a beam of X-rays can be directed almost like a bullet or a knife; (2) because the radiation from radium, like that of the X-rays, diminishes with the square of the distance, which makes treatment with radium at a distance entirely impractical. The distance in the application of radium is measured in millimeters, while most commonly the X-rays are used at a distance of from 20 to 50 centimeters or even 100 centimeters. This is more than 100 times as great a distance,which increases the relative depth value. Therefore, we use radium in deep seated malignant disease, only when it can be inserted into the diseased area, such as in carcinoma of the uterus or when located in some of the cavities, or when radium needles containing the radium emanation or radium element can be distributed evenly throughout the malignant tissue. We use the high voltage X-rays when tumor cells must be destroyed at a depth of more than 2 or 3 centimeters and when crossfiring is an important factor.

Radium must no longer be looked upon as some magical substance which will work miraculous cures when applied to a patient suffering from malignant disease. Radium is an element which in its decomposition produces effects, obeying physical laws which are as definite as the law of gravity or the laws governing the light from the sun. So, too, the X-rays obey definite physical laws which must be understood and utilized properly.

LOW VOLTAGE RAYS VS. HIGH VOLTAGE RAYS

The high voltage rays used in recent years have increased the penetrative value of the radiation about 25 to 30 per cent. Therefore, as I stated several years ago, one would expect about 25 to 30 per cent improvement in our therapeutic results in deep seated malignant disease, and this is about what we are getting. Greater dangers are, however, involved, and greater skill and caution are required. These agents are by no means a "cure-all." There will be more failures than successes. Most will depend

upon early recognition and early and skillful treatment of the disease, either by these agents or operation.

THERAPEUTIC RESULTS

The three great fields in which radiation therapy has been proved of definite value in the treatment of deep seated malignant disease are in carcinoma of the breast, carcinoma of the uterus, and in lymphatic tumors. While some brilliant results have occasionally been obtained in malignant disease of the viscera, for the most part radiation therapy is as unsatisfactory as other methods of treatment of such cases. Therefore I think we will accomplish more in the brief space of time at our command, if we discuss in more detail the above mentioned two or three groups.

CARCINOMA OF THE UTERUS

Most work has been done in malignant disease of the uterus. The results accomplished by the radiologists in the treatment of inoperable and hopeless cases of carcinoma of the uterus have gradually convinced the gynecologists of the value of radiation treatment, and gradually one clinic after another has taken up the radiation treatment and applied it in the borderline and operable cases, until now radiation is the method of choice in the treatment of all cases of carcinoma of the uterus, except possibly in carcinoma of the fundus. Radiation is the method of choice in operable cases only, however, if the proper facilities are at hand, and if sufficient skill and technical knowledge has been developed to give the treatment properly.

Many convincing tables of statistics have been prepared, but the ones covering the widest range, and the longest period of time, I believe, are those from the Doederlein Clinic, in Munich (Table I), prepared by Seuffert (6).

The group of cases making up the operation statistics were treated during the years from 1908 to 1912, while those used for the radiation statistics were those treated from 1913 to 1916. This makes a fair comparison because all classes come into consideration, and since the diagnosis and the classification

TABLE I.—STATISTICS ON CARCINOMA OF THE UTERUS FROM THE DOEDERLEIN CLINIC, MUNICH, AS COMPILED BY SEUFFERT

Therapy	Total Cases in Clinic	Treated Cases	Treated Per cent	Five-year Cures Cases	Five-year Cures Per cent
GROUP I.—Operable					
Operation	265	110	42	51	46
Radiation	500	77	15	37	48
Completed radiation treatment	77	43	55	35	80
GROUP II.—Borderline					
Operation	265	57	22	3	5
Radiation	500	90	19	18	20
Completed radiation treatment	90	50	56	18	36
GROUP III.—Inoperable					
Operation	265	92	34	0	0
Radiation	500	214	43	13	6
Completed radiation treatment	214	121	57	13	11
GROUP IV.—Hopeless?					
Operation	265	6	2	0	0
Radiation	500	119	23	1	0.8
Completed radiation treatment	119	20	17	1	5

TABLE II.—COMPARISON OF RESULTS OBTAINED BY OPERATION AND RADIATION, DOEDERLEIN CLINIC, AS COMPILED BY SEUFFERT.

	Cases	Per Cent	Cured	Per Cent
Cases Treated by Operation—1908 to 1912				
Group I	110	42	51	46
Group II	57	22	3	5
Group III	92	34	0	0
Group IV	6	2	0	0
Groups I and II	167	63	54	32
All groups	265		54	20
Cases Treated by Operation—1913–1916				
Group I	35	46		
Group II	5	5		
Group III	0	0		
Group IV	0	0		
All Groups	40	24	40	8
Cases Treated by Radiation—1913–1916				
Group I	77	15	37	48
Group II	90	19	18	20
Group III	214	43	13	6
Group IV	119	22	1	0.8
Groups I and II	93	55.5		
All groups	500		69	14
Cases Completing Radiation Treatment				
Group I	43	55	35	80
Group II	50	56	18	36
Group III	121	57	13	11
Group IV	20	17	1	0.8
Groups I and II	93	55.5	53	58
All groups	234		67	29

were all made in the same clinic from the same class of patients, true comparison can be made.

In order to make the relative values more clear, Seuffert has determined the absolute values in terms of cures (5 years), in terms of percentage of the total number treated, by operation and those treated by radiation, in values of the total number applying for treatment at the clinic as shown in Table II.

These comparative tables show in the Group I, of clearly operable cases, 46 per cent cured by operation, while 48 per cent were cured by radiation, and of those cases which completed the radiation treatment, 80 per cent were cured. The 80 per cent represented the value to the individual patient *who can complete the treatment.* In Group II, which are the borderline cases, operation cured 5 per cent, while radiation cured 20 per cent, or four times as many, and of those which completed the treatment, 36 per cent were cured. In Group III, which were clearly inoperable, operation gave no cures, and radiation cured 6 per cent. Of those which were completely treated, 11 per cent were cured, as compared with no cures by operation. In Group IV, which were considered absolutely hopeless, none was cured by operation, and one case, or a little less than 1 per cent, was cured by radiation.

In the study of Table II, which shows the absolute value, it is found that of the 265 operated upon during the years 1908 to 1912, 54 cases, or 20 per cent, were cured, but when the same curative values by operation are applied to the group of 500 patients who visited the clinic during 1913 to 1916, only 8 per cent could have been expected to be cured, while of these same 500, 14 per cent were actually cured by radiation, or nearly twice as many. The superiority of the radiation, as applied to all cases, is therefore definitely established.

If one only considers those cases which completed the radiation treatment, 29 per cent of all cases were found cured.

These statistics correspond very closely to those prepared by Heyman (4) from the cases treated in the Radium Home in Stockholm, under the direction of Forssell. In the operable and borderline cases he obtained 40 per cent cures after at least 5 years, by radiation treatment. These results also correspond fairly well to those reported by Greenough, as chairman of the committee on treatment of malignant diseases with radium and X-ray, appointed by the American College of Surgeons, (3) in which 829 cases of carcinoma of the cervix, proved by microscopic examination, and treated by operation, or radiation, or cauterization or by combinations of these methods, were reviewed and analyzed.

Of 829 women with cancer of the cervix, 94 were free from disease 3 years or longer after treatment. More than half of these "cures" were obtained by the use of radium and the X-ray without radical operation. No "cures" were obtained with the cautery alone. In 243 early, favorable, and borderline cases, hysterectomy alone cured 1 in 3, with an operative mortality of 1 in 5. Radium with palliative operation (cautery) cured about 1 in 3, and radium alone (or with palliative operation) about 1 in 5.

In all instances I must urge that one must have a sufficient quantity of radium and must use in the neighborhood of from 5,000 to 7,500 milligram or millicurie hours of gamma radiation and sufficient high voltage rays to destroy the outlying cancer cells. One must have sufficient skill to distribute this radiation evenly throughout the diseased area and yet not overdose the essential organs in the pelvis.

The above discussion applies to "cures." One must not lose sight, however, of the value of radiation as a palliative treatment in the inoperable groups, where one obtains relief from pain, hæmorrhage, and foul discharge. In fact, in these advanced cases, so long as the disease is confined to the pelvis, one may obtain temporary complete relief of all symptoms and the patient may consider herself well.

CARCINOMA OF THE BREAST

The next great group of cases of deep seated malignant disease is carcinoma of the breast. In this group we have very few statistics. Statistics in carcinoma of the breast are most difficult to prepare, because

very few early cases of carcinoma of the breast have been referred for treatment. Nearly all cases have been very advanced and hopelessly inoperable primary cases with recurrences or with metastasis. The other breast cases have been referred for pre-operative or postoperative treatment. I have written in detail upon these subjects and will discuss them only briefly here (5).

Pre-operative radiation is indicated because, as has been shown experimentally, (1) it devitalizes the malignant cells so that they are not easily transplanted, and (2) because tissue that has been irradiated does not easily "take" cancer when implanted, and in fact it has a destructive effect upon cancer cells when implanted, as shown at the Rockefeller Institute.

Postoperative radiation has been used over a longer period of time, and some statistics are appearing as a result. The most convincing of these are those by Anschuetz (1).

The clinical material was obtained from the surgical department at the University of Kiel, consisting of 230 cases of cancer of the breast, operated upon by the same surgeon and verified histologically. All deaths occurring afterward were attributed to cancer, though they may have been due to intercurrent disease. The cases were classified into three groups.

Group 1. Small movable cancers, without palpable axillary lymph nodes.

Group II. Infiltrating cancers, with adhesions and palpable axillary lymph nodes.

Group III. Large infiltrating cancers with axillary and supraclavicular lymph nodes:

TABLE III.—STATISTICS OF ANSCHUETZ AND HELLMAN ON CANCER OF BREAST

	Cases	Per cent Cured
Group I, Series A	7	100
Group I, Series B	6	100
Group II, Series A	103	35
Group II, Series B	96	57
Group III, Series A	8	12.5
Group III, Series B	10	33

Series A: 118 cases operated and not irradiated.
Series B: 112 cases operated and postoperative irradiation.

The value of postoperative irradiation is almost universally recognized. Almost every one has seen the remarkable disappearance of recurrent carcinoma, and since all recurrences develop from retained carcinoma cells, it is logical to assume that the treatment which will make macroscopic lesions disappear should also make microscopical lesions disappear. Therefore, the postoperative radiation should be applied as soon as practical after operation.

I think that in the future more primary cases will be treated by radiation. We have treated a number of primary cases in which the lymph nodes have disappeared and the infiltrating carcinoma has become freely movable. When this local mass is then removed and studied microscopically, at times one finds no evidence of cancer. At other times a few cancer cells can be found embedded in the fibrous tissue. We have also had some excellent results in the treatment of primary cases with no operation.

The radiation treatment of distant metastases following operation is generally followed by retardation and at times disappearance of the lesion treated, but practically always the patient develops other metastatic disease and ultimately dies of carcinoma.

The opinion is becoming pretty definitely fixed that more good is accomplished and less harm done in carcinoma of the breast, by fractional dose treatment (relative) than by an attempt to deliver the full treatment in one day.

Time will not permit a detailed discussion of the treatment of other deep seated malignant disease. Most will be accomplished, however, in the individual case by a conference with the attending physician, the surgeon and the radiologist before any line of treatment is decided upon. At this conference the general condition of the patient, the extent of the disease and its nature should be determined, and then the best means adopted for complete eradication of the disease.

CONCLUSIONS

The following conclusions may be drawn:

1. Patients should be taught to apply early for treatment. Any lump or abnormal bleeding may be due to cancer.

2. Physicians should learn to recognize cancer in its early stages.

3. Pre-operative irradiation will devitalize the cancer cell and prevent its transplantation or dissemination.

4. Postoperative irradiation should destroy remaining carcinoma cells.

5. Thorough and skillful treatment by radiation offers most in all stages of carcinoma of the cervix. Sixty to 80 per cent may be expected to recover if treated in the earliest stages, while less than 1 per cent will recover in the late stages.

6. Radiation will not cure generally disseminated cancer. The more extensive the disease, the less the chance of recovery. Radiation is a local method of treatment.

7. Skill is required in deep radiotherapy in the same sense and degree that is required for successful surgery. Surgical instruments are to the surgeon what radium and the X-rays are to the radiologist.

REFERENCES

1. Anschuetz and Hellman. Muenchen. med. Wchnschr., 1921; Ref. Béclère, La roentgenthérapie préventive post-opératoire du cancer du sein. J. de radiol. et d'electrol., 1924, viii, No. 9.

2. Ewing. The Mutter Lecture before the College of Physicians of Philadelphia, Nov. 1, 1922.

3. Greenough. The treatment of malignant diseases with radium and X-ray—cancer of the cervix. Surg., Gynec. & Obst., 1924, xxxix, 18.

4. Heyman. Technique and results in the treatment of carcinoma of the uterine cervix at "Radiumhemmet," Stockholm. J. Obst. & Gynæc., Brit. Emp., 1924, xxxi, No. 1.

5. Pfahler. Inoperable primary carcinoma of the breast. New York M. J. 1913, April 26. Treatment of recurrences and metastases from carcinoma of the breast. Surg., Gynec. & Obst., 1913, Nov. Deep roentgen-therapy in the postoperative treatment of carcinoma of the breast. Interst. M. J., 1915, xxii. Cancer of the breast, non-operative treatment, diseases of the breast. Deaver and McFarland, Philadelphia: P. Blakeston's Sons & Co., 1916. The treatment of recurrences and metastases from carcinoma of the breast by means of radium and roentgen-rays. Am. J. Roentgenol., 1920, vii, 328–332. Radium combined with X-ray treatment in carcinoma of the breast. Am. J. Roentgenol., 1921, viii, 661–668. Radiotherapy in carcinoma of the breast. Surg., Gynec. & Obst., 1922, Aug. 217–226. Deep roentgen-therapy in the treatment of carcinoma of the breast. Am. J. Roentgenol. and Radium Therapy, 1923, x, 566–568. Radiation treatment of carcinoma of the breast. Atlantic M. J. 1924, Jan. Radiation in the treatment of carcinoma of the breast. South. M. J., 1924, xvii, 203–207.

6. Seuffert. Strahlen-Tiefen-Therapie und ihre Anwendung in der Gynaekologie. Berlin: 1923.

OSTEO-ARTHRITIC PROTRUSION OF THE ACETABULUM

By PHILIP LEWIN, M.D., F.A.C.S., Chicago
Assistant Professor of Orthopedic Surgery, Northwestern University Medical School; Attending Orthopedic Surgeon, Cook County Hospital; Junior Attending Orthopedic Surgeon, St. Luke's Hospital.

BECAUSE of the rarity of osteo-arthritic protrusion of the acetabulum and of its almost complete absence from the American or English medical literature the subject seems worthy of a short discussion with the report of a case.

This condition was first described in 1824 by Otto, of Berlin. In 1921 Valentin and Mueller gave it the name *intrapelvine pfannenvorwoelbung* or Otto-Chrobak pelvis. The only article in English that I have been able to find is that by Hertzler. He states that 34 cases were reported before his 4. The case herein reported brings the total to 39.

The cause of this condition is a combination of factors namely, (1) hypertrophic arthritis of the hip joint; (2) weakening of the acetabular floor; (3) trauma due to weight-bearing or injury; and (4) muscular contraction forcing the head of the femur against the acetabular floor.

The hip is peculiar in that it is a large joint with comparatively small bones and powerful muscles surrounding it. Atmospheric pressure is great.

The pathology is that of (1) arthritis of the hypertrophic type and (2) protrusion of the femoral head through the acetabular floor.

The symptoms are those of a low grade chronic hypertrophic arthritis, namely, limp, pain, sensitiveness, tenderness, and limitation of motion in all directions but especially abduction and external rotation.

The roentgen-ray findings are those of hypertrophic arthritis producing a cap-like shell of bone surrounding the femoral head and the protrusion of the head through the acetabular floor projecting into the pelvis.

The direct diagnosis is made on the findings enumerated above. The differential diagnosis lies between an ordinary osteo-arthritis and Charcot hip. The roentgenogram should determine the diagnosis.

The prognosis is bad as far as motion is concerned, but good as regards relief from pain, as ankylosis occurs.

The course is long.

Treatment consists in search for and removal of foci of infection followed by orthopedic treatment. This includes (1) absolute rest in bed; (2) application of Buck's extension and abduction weight and pulley traction; (3) elevation of the foot of the bed, about 8 inches; (4) local anodyne applications to the affected hip region; (5) application of a plaster of Paris spica cast or of an abduction hip brace; (6) 2 crutches and a 2 inch block under the heel and sole of the shoe of the opposite side. The cast should be worn about 8 weeks and the hip then treated by diathermy and massage.

J. S., white male, 64 years of age, entered St. Luke's Hospital as a private patient, January 14, 1924, complaining of pain and stiffness in his left hip.

Present complaint. There was pain in the left hip joint in all positions of the limb, and rather acute pain at times, but always a dull ache, made worse by walking, and not relieved by sitting.

Onset and course. Eighteen years ago he fell 12 feet down an elevator shaft, landing on his feet. It was necessary for him to go to bed for a short time. Since then he states that the hip has bothered him. gradually growing worse until now, when he can scarcely walk because of pain. He is especially troubled after he has been sitting down for a time and attempts to rise. His hip is then very stiff and painful and after walking a short distance seems to limber up somewhat. However, he cannot walk very far without suffering fatigue of the left leg. The hip has never been swollen or tender. No other joints have ever been involved.

Past history. He has not been ill in any way for a number of years. He has had no tonsillitis or colds, but is subject to catarrhal disease of the nose.

No operations have been performed, and there have been no injuries or accidents other than those mentioned. There was a possible specific urethritis 20 years ago, but chancre is denied.

Inventory by system was negative except for nocturia 1 to 4 times, depending upon the amount of water taken.

Family history was unimportant.

Habits. The appetite was good; sleep was restless because of the hip; the bowels were normal.

The patient smoked 10 to 15 cigars daily until 1 year ago and then stopped. He did not use alcohol.

Fig. 1. Roentgenogram of hip showing osteo-arthritic rim of new bone formation and protrusion of head of femur through the floor of the acetabulum.

Physical examination. Patient is a well developed robust man of 64, not acutely ill, with no irregularities or tenderness of scalp or skull. He is nearly bald. The pupils are equal and react to light and accommodation; they are both slightly flattened in the superior and nasal quadrant. There are no abnormal eye signs and the vision is not impaired to gross tests. The hearing is not grossly impaired. There is no discharge, deformity, or other abnormality of the ears. The nose shows no deformity, obstruction, or discharge. The tongue is clean and protrudes in the mid-line without tremor. The teeth are all false. No roots remain. In the throat are no areas of inflammation. The tonsillar crypts are clear. There is no stiffness or adenopathy of the neck. The thorax is well muscled, broad, and has normal excursions. The heart is not enlarged. There are no murmurs or thrills. The sounds are muffled but regular. The lungs show normal resonance. There are no abnormal voice or breath sounds or tactile fremitus. No râles are heard.

The abdomen is scaphoid, and the muscles well developed. There are no masses, tenderness, spasm, distention, etc. The liver and spleen are not palpable. The genitalia are normal, with no scars or discharge.

The right leg and hip are normal in all respects. The knee jerks are equal and active. There is no Babinski or ankle clonus. There is limitation of motion and muscle spasm about the left hip joint, but no notable swelling, heat, or redness. The joint cannot be flexed past a right angle, and cannot be hyperextended. Abduction and rotation are limited.

Progress notes. January 15, 1924. The patient entered the hospital yesterday complaining of pain in the left hip. Anodyne lotion and hot fomentations were ordered and traction applied. Foot of bed was elevated 8 inches.

Roentgen findings. "Left hip: The acetabulum is greatly thickened, the condition having the appearance of an osteosclerosis. The articular surface of the acetabulum seems eburnated. The head of the left femur is thickened, and I believe is displaced inward. There is, however, no evidence of a fracture involving the proximal third of the left femur. The increased density involving the acetabulum may be due to old injury, accompanied by an infection, which is in a stage of repair."

January 17. There is no complaint. There has been no further pain in the hip. Cast will be applied in a few days.

January 21. There has been no pain in the joint since treatment was begun. Crutches are ordered.

January 23. Traction is removed and spica cast applied.

January 25. There is no pain in the hip.

Patient is discharged in good condition, using 2 crutches and a 2 inch block under right heel and sole. He is to wear the cast about 8 weeks and follow its removal with diathermy and massage.

NOTE—The roentgenogram and the roentgen findings reported in this article are the work of Dr. E. L. Jenkinson.

BIBLIOGRAPHY

1. CHIARI. Beitr. z. klin. Chir., 1916, cii.
2. EPPINGER. Beitr. z. Geburtsh. u. Gynaek., 1903, ii, 176.
3. ESAU, P. Deutsche Ztschr. f. Chir., 1908, xci, 611.
4. FERE. Arch. de neurol., 1882, iv.
5. HENSCHEN. Beitr. z. klin. Chir., 1909, lxv, 599; 1909, lxii, 294.
6. Idem. Ztschr. f. orthop. Chir., 1913, xxxiii, 438.
7. HERTZLER. Arch. Surg., 1922, v, 691.
8. KIENBOECK. Fortschr. a. d. Geb. d. Roentgenstrahlen, 1912, xviii, 280.
9. KULIGA. Beitr. z. path. Anat. u. z. allg. Path. Festschr. f. Arnold, 1905.
10. OTTO. Pfannenbeckenmiszstaltung infolge deformierender Osteoarthritis. Seltene Beobachtungen zur Anatomie, Physiologie und Pathologie gehoerig, ed. 2, Berlin, 1824.
11. SCHERTLIN. Beitr. z. klin. Chir., 1910, lxxi, 406.
12. SCHLAGENHAUFER, F. Zentralbl. f. Gynaek., 1909, xxx, 228.
13. SENFTLEBEN. Arch. f. klin. Chir., 1860, i, 81.
14. THOMSEN. Arch. f. klin. Chir., 1872, xiii, 235.
15. TRENDELENBURG. Verhandl. d. deutsch. Gesellsch. f. Chir., 1881, x, 61.
16. VALENTIN, B., and MUELLER, H. Arch. f. klin. Chir., 1921, cxvii, 523.
17. WOLFSOHN and BRANDENSTEIN. Arch. f. klin. Chir., 1911, xcvi, 656.
18. WREDE. 82. Versamml. d. deutsch. Naturf. u. Aerzte in Koenigsberg, 1910.

DELTOID PARALYSIS FOLLOWING SHOULDER INJURIES[1]

By JOSEPH F. SMITH, M.D., F.A.C.S., and H. H. CHRISTENSEN, M.D., Wausau, Wisconsin

FOR many years works on anatomy and surgery have contained frequent references to paralysis of the deltoid muscle as a consequence of various types of injuries to the shoulder. In spite of the fact that these observations have been repeatedly recorded, we have searched in vain for any adequate or definite description of the mechanism involved in the production of these injuries or of their exact pathological anatomy. In this connection the following two cases which have recently come under our observation have been of unusual interest to us, and we have thought that a brief report might be the means of eliciting discussion which would throw some light on the obscure mechanism and pathological anatomy of these lesions.

Case 1. H. A., Finlander, laborer, age 36. On November 17, 1923, while cutting timber for a lumber company he was struck on the head and left shoulder by the limb of a falling tree; he was knocked down and remained unconscious, he thinks, for a few minutes. He was cared for by a nearby physician and sent to a hospital in a neighboring town where he remained in bed 2 weeks. He does not know what was done to him by the physician who rendered first aid. He came into our hands on December 3, 1923, at which time he was unable to raise the left arm to a horizontal position. The left deltoid was markedly atrophied and showed the reaction of degeneration. The area of anæsthesia extended over the deltoid area with slightly impaired anæsthesia over a somewhat larger area. This patient showed complete paralysis of the triceps muscle and there was some atrophy of the spinati; the other muscle of the shoulder and arm were intact. The patient was put upon electrical stimulation, massage and passive motions which were continued until March 18, 1924, at which time, as no definite improvement had taken place, it was decided to carry out an operation with a view to locating the site of the injury and of repairing it, if possible.

On account of the possibility of paralysis of the spinati and the definite paralysis of the triceps, we first cut down upon the primary divisions of the plexus in the neck and were able to demonstrate definitely that the first, second, and third primary divisions were intact and free. An incision was then made along the anterior border of the axilla after the method described by Stookey, the pectoralis major cut near its insertion, the neurovascular bundle exposed and followed from below upward. The circumflex nerve was found by following upward the musculospiral nerve to the origin of the circumflex as it springs from the musculospiral and enters the quadrilateral space just above the latissimus dorsi tendon. On freeing the nerve at a distance of about an inch from its origin, we came upon a definite bulbshaped neuroma beyond which the continuity of the nerve was severed. The neuroma was resected and what was thought to be the peripheral fibers of the nerve were freshened and brought up and sutured to the proximal segment. The cut muscles were sutured and the wound closed. The patient was seen 9 months after the operation at which time he showed little, if any, signs of nerve regeneration.

Just what happened to this man at the time of the injury we could not ascertain. It is possible he may have had a dislocation which was reduced by the physician who rendered first aid. The point of interest in this case is the finding of a definite solution of continuity in the circumflex nerve with the development of a typical bulbous neuroma at the site of section.

Case 2. H. Y., laborer, aged 48. injured April 11, 1924, when he was struck on the left side of the head and on the left shoulder by a falling limb. He was knocked down and dazed but not rendered uncon-

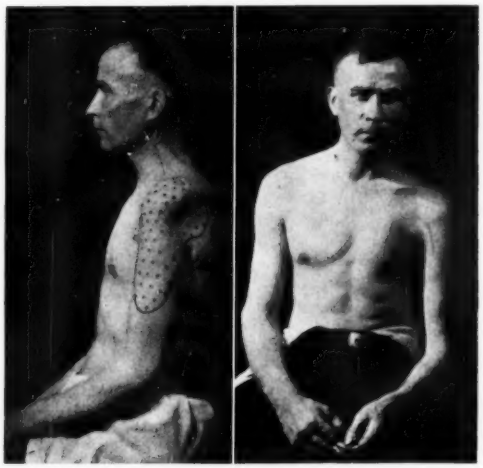


Fig. 1 (left). Case 1, showing area of anæsthesia.
Fig. 2. Incision for exposure of circumflex curve, Case 1.

[1] Read before the Chicago Surgical Society at the regular meeting February 6, 1925.

Fig. 3. Area of anæsthesia, Case 2.

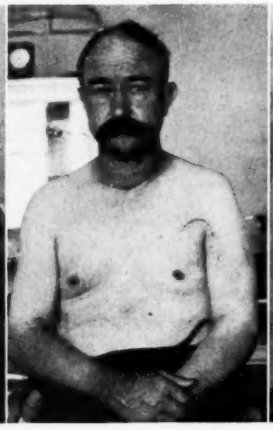


Fig. 4. Anterior incision, Case 2.

Fig. 5. Posterior incision, Case 2.

scious. He says his left arm was turned inward and that he was unable to move it until he was helped up and a pull made on the arm when the shoulder "cracked" following which he could bring the arm in front of the body and hold it up with the other hand. Two weeks later his arm was manipulated and he "felt something slip in the shoulder" after which his shoulder felt better. The patient says he was was later etherized and the arm manipulated.

On admission May 12, 1924, the patient was unable to abduct the left arm; there was some general lameness of the whole shoulder, arm, and hand. All motions of the arm, forearm, and hand were present except abduction of the arm. Examination showed no voluntary power in the deltoid and there was some flattening of the shoulder but no tendency of the humerus to fall away from the glenoid. The reaction of degeneration was present in the deltoid and there was anæsthesia to touch and pain over the deltoid region. All the other muscles of the shoulder, arm, and forearm responded to faradic stimulation. The patient was treated expectantly as in the other case until Nobember 8, 1924, at which time, as there was no definite improvement operation was decided upon and carried out.

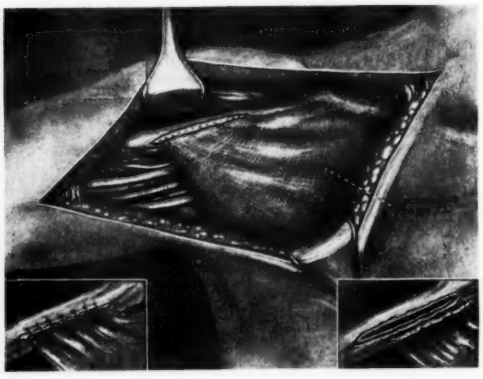


Fig. 6. Drawing showing reflection of pectoralis major muscle. (After Stookey.)

In this case, the quadrilateral space was first exposed posteriorly by an incision along the posterior border of the deltoid. The nerve branches to the muscle were then exposed and found to be intact. An incision similar to that described in the first case was then made and the nerve exposed anteriorly as it enters the quadrilateral space. About one-half inch from its origin from the musculospiral nerve, it was found replaced by a thin scar which was excised to the point where nerve fasciculi were visible and end-to-end suture made. The wound was closed in the usual manner. Up to the present time there has apparently been slight return of function but it is still too indefinite to enable one to predict the probable final result.

In this case again there was a definite anatomical lesion of the circumflex nerve resulting from an injury, which in all probability was primarily a subcoracoid dislocation of the shoulder joint.

In neither of these cases was there any X-ray evidence of bony injury at the time the patients came into our hands.

Tees in the *Canadian Medical Journal* of January, 1921, says that rupture of the circumflex nerve causes a severe paralysis of the

deltoid, later partly compensated by the pectoralis major, supraspinatus, trapezius and serratus magnus. Bunts in the *Transactions of the American Surgical Association* in 1903 reviewed the history of nerve injuries about the shoulder joint and says that Galen in his works refers to a description of this condition as a complication of luxations of the shoulder joint.

Injury to this nerve in civil life results from cuts or other subcutaneous injuries, as a sequel to dislocations of the shoulder or falls upon the shoulder, rarely to crutch or miner's paralysis. Tees has found but few cases reported. He reports one case of his own of a shrapnel injury to the nerve which resulted in the typical disability and did not yield to conservative treatment. He operated upon this case, making a posterior incision and finding the nerve ruptured at the point where it branches before entering the muscle. He tied the three branches together and sutured them to the vivified proximal portion. Five weeks after the operation, Tees says, the power of the deltoid had increased so that the patient could hold the arm horizontally when standing and could abduct the arm to a right angle with the body when recumbent.

Benisty in several hundred cases of peripheral nerve lesions has not seen a case of isolated paralysis of the circumflex nerve and thinks that this lesion is always associated with paralysis of other collateral or terminal branches of the cervical or brachial plexus.

Sherren says injuries of the circumflex are by no means as common as the textbooks would lead us to believe and thinks most cases of deltoid paralysis following injury are due to a lesion of the fibers of this nerve as they run in the fifth cervical primary division and are usually accompanied by paralysis of the spinati muscles. He says that an injury to this nerve causes well defined symptoms. The deltoid is wasted and the acromium is prominent but the head of the humerus does not tend to fall away from the glenoid cavity as it does when the spinati are affected in addition to the deltoid. He points out that sensory changes always accompany injuries to this nerve, complete division resulting in loss of epicritic and protopathic sensibility over an area corresponding to the deltoid muscle. He lays down the rule that paralysis of the deltoid without sensory change is due to interference with the function of the fifth cervical nerve and not with the circumflex.

SUMMARY

Herewith are presented two case histories of non-penetrating injury to the shoulder resulting in paralysis of the deltoid both showing the typical area of anæsthesia and both showing no tendency of the humerus to fall away from the glenoid. One case was uncomplicated by any other nerve injury and in the other case there was a concomitant injury to the branches of the radial supplying the triceps muscle but with no demonstrable lesion of the first three primary divisions of the plexus. In this (first) case, we have assumed that in all probability we had to deal with two peripheral nerve injuries; namely, that of the circumflex resulting in deltoid paralysis and of the muscular branches of the radial supplying the triceps. In both cases, a definite lesion (solution of continuity) of the circumflex nerve was demonstrated at operation. In both cases, there was no X-ray evidence of bone injury at the time they came into our hands. The exact mechanism of injury was also obscure but at least in one case was probably due to a subglenoid dislocation of the upper end of the humerus.

CONCLUSION

These two cases apparently demonstrate that even common injuries of the shoulder without crushing, penetration, or laceration may result in definite solution of continuity of the circumflex nerve with resulting complete and permanent paralysis of the deltoid muscle.

REFERENCES

BENISTY. Clinical Forms of Nerve Lesions.
BUNTS. Tr. Am. Surg. Ass., 1903.
CUNNINGHAM. Textbook of Anatomy.
TEES. Canadian M. J., 1921, Jan.
SHERREN. Injuries of Nerve, 1908.

DIABETIC AND NON-DIABETIC GLYCOSURIA IN THE SURGICAL PATIENT

By WILLIAM C. MENNINGER, M.D., Topeka, Kansas
From the Second Surgical (Cornell) Division, Bellevue Hospital, New York City

IT is our intention to present our experience in the care of surgical patients showing glycosuria, not only diabetic but non-diabetic glycosuria. The former group usually presents the more serious problem and will receive the major consideration, while the latter group which includes cases which may require special intensive treatment, will be mentioned briefly.

OCCURRENCE AND MORTALITY

The frequency of glycosuria in surgical patients shows a much larger figure than the frequency of surgical complications in the diabetic. No definite figures can be given for the former; of the latter, Joslin (37) found 11 per cent of 903 cases of diabetes mellitus required surgical treatment and Fitz (25) reports 14 per cent of 380 cases. The frequency of trauma, which would class the patient as surgical, as a factor in diabetes was found by Diez (21) to be 2 per cent in a series of 4,068 cases, and 5.6 per cent in a second series of 669 cases; Cantani (11) found a history of trauma in 10.25 per cent of 1,004 cases; Griesinger (31) 5.7 per cent in 225 cases; Frerichs (28) 2 per cent of 400 cases; Seegen (55) 1.4 per cent of 938 cases; Kuelz (44) 5.6 per cent of 692 cases; and Ebstein (22) 1 per cent in 116 cases. The occurrence of glycosuria following trauma is much more frequent. In head injuries of varying severity, Higgins and Ogden (33) found glycosuria in 9.43 per cent of 212 cases. Konjetzny and Weiland (42) noted 40.9 per cent of 83 cases of fractures of the extremities showed a spontaneous glycosuria of a temporary character, associated with polyuria, polydipsia, and hyperglycæmia. The occurrence of glycosuria following an operation in the non-diabetic is generally known and careful study of the blood sugar pre-operative and post-operative has recently been made by Chantraine (12) and Dewes (20), the latter noting that in the great majority of laparotomies, a hyperglycæmia from two to four times the normal figure resulted.

The old dictum of Verneuil (64) that "the presence of glucose in the urine, whatever its amount, is always a serious fact," remains disconcertingly true. However, the most frequent serious glycosuria which confronts the surgeon occurs with diabetes mellitus. The distinction between a surgical condition complicated by diabetes and diabetes complicated by surgical interference makes little difference, since in either case, the immediate responsibility rests with the surgeon. The mortality previous to the use of insulin as given by various writers varies widely, due to the reference of some to mild or moderate cases and of others to severe cases. Banting, Campbell, and Fletcher (5) refer to a 70 per cent mortality up to 1914; Foster (26) states that 60 per cent of fatalities in diabetics over 30 years of age occurs following operations; Fitz (25) found 50 per cent mortality in infected or gangrene cases, and 12 per cent in non-infected cases; Gardner (29) gives an 80 per cent mortality; Young (67) found 16 per cent in 99 cases; Krecke (43) found 30 per cent in 20 cases; Berkman (6) found 9.5 per cent of 159 cases, many of which were mild; Karewski (39) found 11.8 per cent of 68 cases died in coma following operations on aseptic tissues and 21.7 per cent of 69 cases after operations on infected tissues; in Phillips' (51) summary, there was a 28 per cent mortality; Joslin (37) prior to 1917 found 18 per cent mortality in 27 cases, not including sepsis or gangrene; in the 77 cases of diabetic gangrene amputation given by Joslin, Binney (7), Jones (36), and Young (67), there was a 48 per cent mortality; in 17 cases of gangrene, Stetton (58) noted 29 per cent mortality; Weeden (66) notes 36.8 per cent in 160 cases; Strouse (59) found 31 per cent in 38 operated cases. In the present series which includes 47 cases from 1916 to

1922, the large majority of which had moderate or severe diabetes, there were twenty known deaths and four probable deaths, the latter leaving the hospital in a severe condition, refusing all treatment. Omitting these four cases, the mortality of this series was 42.5 per cent.

Since the advent of insulin, fewer figures are available. Allen and Sherrill (3) found that among 14 cases complicated by local or general infection, there were five deaths. Weeden (66) reports a mortality of 16.6 per cent in a series of 12 cases, three of which as judged from their clinical course, owe their lives to insulin treatment. Joslin found a 9 per cent mortality in a series of 61 cases omitting carbuncles and gangrene. Since 1923, 22 cases of diabetes mellitus associated with some surgical condition have been treated on the Second Surgical Division at Bellevue, all but two of which received insulin, and with but one death (4.5 per cent). Of this number, five would have had very little hope without insulin. The one death was in an extensive carbuncle of the neck, although the diabetes responded to insulin.

Carbuncles have long been associated with diabetes, as a result, according to Higginson (34), of the "sapræmic poisoning" from the carbuncle lowering the saturation point for the body, with hyperglycæmia or glycosuria resulting. Because of the difficulties in treatment, the mortality is high. Karewski (39) reports a 33 per cent mortality in 37 cases. In 42 carbuncle cases reported by Muller (48), 6 (14.3 per cent) were diabetic, of which 4 (66.7 per cent) died while only one (2.8 per cent) death occurred in the non-diabetic group. Since 1916, there have been 123 cases classed as carbuncles treated on the Cornell Service of Bellevue, 13 (10.5 per cent) of which occurred in diabetics. Of the 13 diabetics, there were 7 deaths (53.8 per cent), while there were only 7 (6.3 per cent) deaths in the 110 non-diabetics. As Muller has pointed out, this is sufficient evidence to disprove the older opinion as expressed by Smith and Durham (56) that the presence of glycosuria does not have a tendency to influence the course of the carbuncle in any way for the worse.

ANÆSTHESIA

The question of anæsthesia in operations on diabetics is a moot point and has received a great deal of discussion. Nearly every possible anæsthesia has at some time been advocated by someone and advised against by others. Local anæsthesia is nearly universally advocated either alone (Bruce, 10; Plicque, 52; Labbe, 45; Umber, 63) or with nitrous oxide if necessary (Murphy, 49; Tytgat, 62; Leyton, 46; Kahn, 38) or with ether (Jones, McKittrick, and Sisco, 35; Adams and Wilder, 1). Muller (48) feels it is contra-indicated since it predisposes to extensive necrosis if infection occurs. Procain, novocain, and cocain are most commonly used. Nitrous oxide probably has more advocates than any other form on the principle that the patient recovers more quickly and the alkaline reserve is less affected (Berkman, 6; Costain, 15; Fitz, 25; Murphy, 49; Leyton, 46; Cheever, 14; Cruikshank, 16; Jones, 36; Kahn, 38; Young, 67; Muller, 48). The use of ether instead of nitrous oxide is advocated by a few (Jones et al, 35; Adams and Wilder, 1). Ether is strongly advised against by Labbe (45), Jones (36), and Muller (48), and Harrop (32) states that it should be avoided since it has recently been shown that it probably has a specific destructive action on insulin. Spinal anæsthesia is advocated by Labbe (45), Leyton (46), Umber (63), and Muller (48). Ethyl chloride has been successful with Labbe (45) and Plicque (52). Chloroform has been used by Chavannez (13) unsuccessfully and is generally considered the poorest anæsthetic (Murphy, 49; Plicque, 52; Labbe, 45; Cruikshank, 16; Jones, 36; Blum, 8; Farr, 23).

An interesting sidelight on the question is thrown by Dewes (20) and also Chantraine (12). Dewes found that in laparotomies, there was an increase in the blood sugar with either local anæsthesia or ether to two to four times the normal figure, and with no difference between the two forms of anæsthesia.

On the Second Surgical Division, the anæsthetic varies with the case. Spinal anæsthesia has been used successfully in leg amputations. For short minor operations (carbuncle, abscess, cellulitis) gas-oxygen with local anæsthesia has been very satisfac-

tory, as also has been ethyl chloride. In one case of multiple abscesses, ethyl chloride was administered five times and has proved probably the most satisfactory for short operations. For more prolonged operations (cholecystectomy, mastectomy), we use gas-oxygen, changing to gas-oxygen-ether mixture after induction has been well started.

TREATMENT

The treatment may be divided into preoperative and postoperative, but every case of diabetes in surgery requires treatment somewhat different from any other case, and while specific methods as used on this service cannot be outlined in detail, an attempt will be made to list the general steps. The basic principles on which the treatment is given are (1) to provide a maintenance diet and (2) to control the diabetes with insulin as early as possible, maintaining the blood sugar at about the renal threshold, controlling the dosage by frequent urinalysis and where necessary blood sugar tests. We feel that there is a marked contrast between the way uncomplicated diabetes and diabetes complicated with surgical interference should be treated. Time is of tremendous importance in surgery in even moderate diabetics, and generally cannot be spent, except with much increased danger to the patient, in waiting several hours or longer for a blood sugar test. "Surgical" diabetes *must* be treated intensively at the earliest possible moment until the surgical condition is remedied; a point which we feel is not always fully appreciated by the internist.

Whether it is before or after operation, with the patient in coma or with mild diabetes, the immediate problem is the same, namely, to gain control of the diabetes. In severe cases requiring operation, where marked acidosis is present, if an interval of 12 hours or more can be spared during which intensive treatment can be given, the chances for recovery from the surgical condition are much improved.

There are certain general steps followed out in every case of diabetes.

1. *Urinalysis*, including specific gravity, amount, qualitative sugar, acetone, and diacetic acid, is the first step in every case, and is performed immediately on admission. The urinalysis is repeated every 3 hours as long as the patient shows sugar. Brigham (9) advises urinalysis of all specimens, but we have found it necessary to catheterize in some cases, rather than wait for voluntary voiding. Benedict's solution is used for testing sugar and the result determines the subsequent insulin dosage as given below. We do not do quantitative examinations on each specimen (although this was tried for a period) since the additional information gained is negligible. As long as the patient continues to show sugar, 24 hour specimens are not saved, but each specimen is examined immediately. Jones, McKittrick, and Sisco (35) follow the plan of frequent urinalysis, but it is performed by the nurse who also administers the insulin dosage according to her findings. On the Cornell service, the urinalysis and insulin dosage is always in charge of the surgeon, and it has been found more satisfactory to have one man assigned to all the diabetic cases on the service.

Of the various tests, the sugar test is the most important. Using Benedict's solution, one can roughly estimate the amount of sugar present by the degree of precipitation of the copper oxide. Diacetic acid is of more significance, if present, than acetone and is less likely to be present on a starvation diet than acetone. However, we differ from Manges (47) who states: "There is no danger no matter how intense the acetone reaction may be, when the diacetic or oxybutyric acid tests are negative." Several of our cases have had a low carbon dioxide content of the blood with 4+ acetone and negative diacetic acid test of the urine. Hence consideration is given to the acetone reaction, and following the clearing of the glycosuria, close attention is paid to clearing the ketosis. In one case (cholecystectomy), there was only a slight amount of sugar after operation, but a marked ketosis which cleared with insulin therapy. Thalhimer (61), Fisher and Snell (24), and Speese (57) have advocated the use of insulin in nondiabetic acidosis.

2. *Insulin administration* follows the urinalysis. Our aim has been to reduce the glycosuria as soon as possible. This is done

by urinalysis every 3 hours, with immediate insulin administration based on the findings at each examination. This 3 hour repetition is continued until the glycosuria is reduced to a trace. As long as the patient has an acute surgical condition, we have found it the lesser of two evils to reduce the glycosuria to a trace, maintaining it about the renal threshold, and so prevent a hypoglycæmia. This is against the advice of Allen and Sherrill (3), and Joslin (37), although the latter was not speaking specifically of surgical conditions, but we are supported by Foster (26), Banting, Campbell, and Fletcher (5), Jones et al (35). Foster has termed it the "buffer sugar."

The quantity of insulin to be given in each dose must be determined by the urine findings (or blood findings), and is largely a matter of experience. On this service, when the Benedict's solution is turned a golden yellow-red, 20 to 25 units are given. The amount is lessened as the solution becomes green. The amount which may be given in any definite length of time certainly has a limit, but as long as glycosuria is present, there is no danger from hypoglycæmia. In one case, 140 units were given in 14 hours, and in a second, 125 in 24 hours with satisfactory results.

In cases where acidosis is present, without glycosuria, or where the glycosuria has cleared, it is necessary to administer glucose with the insulin. At least 50 to 100 grams of carbohydrate should be given a day until the acidosis has cleared. Orange juice by mouth is sufficient or if the patient cannot take fluid by mouth, 5 per cent glucose is given by rectum. If this cannot be retained, 10 per cent or even 20 per cent glucose solution can be given intravenously. In such cases, it has been found advantageous to disregard any glycosuria that may appear until the acidosis is cleared.

One other point has been observed regarding the dosage of insulin which has previously been noted by others. The insulin tolerance in cases of infection is much reduced as the infection subsides. Not infrequently, its administration may eventually be entirely unnecessary. Hence while the initial 24 hour quantity, and often that for the second and third day may be large, it must gradually be reduced. When the glycosuria has cleared and insulin is necessary to maintain the patient's status *pro tempore*, it is given three times a day. We have found Allen's (4) suggestion successful in giving the morning dose about an hour before breakfast, the noon dose a half-hour before or just at meal time, and the evening dose from one-half to an hour after supper, omitting any night dose.

3. *Fluid intake.* Before operation, the patient is given considerable water, on the principle that it is more advisable to give the patient considerable water before operation than to find it necessary to force it after operation, as Joslin (37) and Kahn (38) have suggested. No set amount is given, but the patient is supplied freely by mouth, and if necessary by rectum or subcutaneously. Following operation, the patient receives at least 2,000 cubic centimeters during the first 24 hours, by mouth if possible, or if not, four to six ounces of tap water every 6 hours by rectum, and the rest by saline hypodermoclysis. A 3 per cent glucose saline clysis has been used satisfactorily in indicated cases.

4. *Diet.* We believe that the particular type of diet makes little difference, so long as it is closely watched. As Foster (26) has suggested, and as is used on the Second Medical (Cornell) Division of Bellevue, the patient is started with a quart of whole milk per day. This places the patient immediately on a known food intake, which is continued until a special diet can be obtained. The diet consists of 1 gram of protein per kilogram of body weight, the remainder of the caloric requirement of carbohydrate and fat in a 1 to 3 ratio, allowing 30 calories per kilo of body weight. Petty (50) also uses this diet. The caloric intake is increased to 35 calories per kilo when the patient is allowed out of bed, and to 40 when up and around the ward, depending somewhat on the diabetic condition at the time.

The question of under nutrition is undecided. Allen and Sherrill (3) favor moderate under nutrition. Joslin (37) suggests a preoperative dose of 15 to 20 calories per kilogram. Jones et al (35) give 20 calories per kilogram. Delbet (18) feels that carbohydrate need not be decreased. Muller (48) brings the

patient only to the carbohydrate tolerance. Banting, Campbell, and Fletcher (5) increase the carbohydrate above the patient's tolerance and give insulin to prevent glycosuria.

Postoperative specific measures used vary considerably with the operation. The maintenance diet, mentioned above, is reached as soon as possible. In severe cases, only carbohydrate is given the first day; at least 100 grams in 24 hours, in the form of 3 per cent hypodermoclysis, 5 per cent glucose by rectum, or by mouth as orange juice if possible. Such is repeated until danger from acidosis is past. Milk and eggs are added by the third or fourth day even in major operations, followed by a portion of the regular dietary ration, and increasing to the full diet in 6 to 8 days.

5. *Blood chemistry.* While blood chemistry is interesting and may be helpful, in cases other than coma or partial urinary retention, as long as glycosuria is present, it is not essential. It is often impossible and generally impracticable to obtain blood sugar determinations every 3 hours during the active glycosuria reduction. Some workers (19, 36) feel that insulin cannot be given intelligently without knowing the blood sugar, with which we disagree, provided the above conditions are present. As has been mentioned, small amounts of glucose in the urine may even be desirable to aid in the administration of insulin during the acute stage of the surgical condition.

However, in coma, or with partial urinary retention, and when the glycosuria disappears, blood sugar determinations must be carried out to administer insulin intelligently. Also in the presence of nephritis or in other cases, where the threshold is very high, blood sugar determinations are very desirable. One case in this series showed a renal threshold for glucose between 240 and 260 milligrams per 100 cubic centimeters. We do believe that as soon as safely possible the blood sugar should be reduced to and maintained within normal limits.

6. *Alkali and drug therapy.* For many years, alkali has been one of the important therapeutic agents used in diabetes. In the presence of surgical complications, it has been advocated by many (Berkman, 6; Bruce, 10; Koerte, 41; Schwartz, 54; Plicque, 52; Labbe, 45; Umber, 63; Addis, 2; Reisman, 53; Krecke, 43; Muller, 48) but usually in widely varying doses, in various methods of and times for administration. Among medical men, von Noorden and Woodyatt, Allen, Bock, and Fitz (65), have advocated it, but they also vary in their dosage. Foster (27) has found the result often disappointing. Joslin (37) does not use them and found his cases got along just as well or better. In the present series, it was used in the earlier cases, but without observed beneficial effect. It has not been used in the more recent cases.

Other drugs have been used for symptomatic treatment. Cumston (17) suggests strychnine as a tonic to the gastro-intestinal system, glycerin to counteract weight loss, and opium to check thirst. Murphy (49) also mentions opium as a valuable adjunct.

7. The *reduction of the toxæmia* is important in every surgical diabetic. Infection plus the fever gives an increased metabolism. The toxæmia from the infection gives anorexia plus nausea and vomiting, and hence energy must be derived from the body tissues, chiefly from the surplus body fat. The glycogen reserve is at once exhausted and the only carbohydrate comes from the protein and is inadequate for the combustion of the fat. The patient promptly develops a severe acidosis and tends toward an acidosis state as soon as he is infected, for he then burns more carbohydrate than before. The reduction of the toxæmia in many cases depends on immediate and adequate drainage. Insulin, while of tremendous aid, cannot overcome a severe infection (Strouse and Schultz, 60).

TRAUMATIC GLYCOSURIA

The frequency of traumatic glycosuria has been mentioned, but rarely does it prove a serious problem for the surgeon. Kausch (40), in an excellent review of cases up to 1904, is skeptical as to any relation between trauma as a cause and diabetes. Joslin has pointed out that the World War did not increase diabetes. Of the 212 cases studied by Higgins and Ogden (33), only three showed any lasting glycosuria. One case studied by Konjetzny and Weiland (42) showed a blood sugar of 357

milligrams per 100 cubic centimeters. A case reported by Ginsberg (30) developed a blood sugar of 325 milligrams per 100 cubic centimeters following trauma, with acetone and diacetic acid in the urine, which responded well to insulin and entirely cleared in 20 hours. A similar case was admitted to the Second Surgical Division last fall.

M. S., a male of 35, with an entirely negative history for diabetes, was admitted about 3 p.m., having fallen from a scaffolding, striking his jaw and arm. He appeared in good condition, complaining only of pain where struck. X-ray examination revealed no fracture. About 6 hours later, the patient became restless, anxious, and appeared as in shock, suggestive of an internal hæmorrhage. Stimulative measures were given without changing his condition. His urine examined at 11 p.m. showed a heavy precipitate of glucose (Benedict), acetone 2 plus, and diacetic acid. He was given intensive treatment with insulin, receiving 95 units (U20-Lilly) in 24 hours. Clinically he responded rapidly; the urine became entirely negative. He was placed on a maintenance diet of 1830 calories without insulin. Two days later his blood sugar was 120 milligrams and carbon dioxide 50 volumes per cent. He left the hospital on the sixth day, feeling well, was on a regular diet, and showed no symptoms or signs of diabetes.

Although this division has a very active traumatic service, this is the only case of traumatic glycosuria receiving insulin. We feel that this case represents a true diabetes, though temporary, in the sense that glucose is improperly burned or that excessive glycogen is discharged from the liver, with hyperglycæmia and acetone body formation, probably due to a temporary disturbance in the nervous mechanism controlling carbohydrate metabolism. Its treatment is essentially the same as that of a severe diabetic.

SUMMARY

1. In a series of 47 cases treated before insulin, there were 20 deaths (42.5 per cent) and four additional cases which left the hospital in a critical condition refusing treatment. In a series of 22 cases treated with insulin, there was one death (4.5 per cent).

2. Urinalysis is performed every 3 hours as long as the patient shows glycosuria, with insulin administration based on the findings.

3. Fluid intake is encouraged pre-operatively and forced to at least 2,000 cubic centimeters daily postoperatively.

4. The diet given allows 30 calories per kilo body weight, with one gram of protein per kilogram of body weight, and the remainder in carbohydrate and fat in the ratio of 1 to 3.

5. Blood chemistry is very desirable in cases of coma, partial urinary retention, and after glycosuria has disappeared. In the great majority of cases, it is not essential while the patient shows glycosuria and very valuable time is lost, with much increased danger to the life of the patient, in waiting several hours for a blood sugar determination.

6. Alkaline therapy is not used at present.

7. Traumatic glycosuria may be effectively treated, when necessary, with insulin.

I wish to express to Dr. H. E. Santee, director of the Second Surgical Division, my appreciation of his courtesy in affording me the opportunity of reporting the above cases.

REFERENCES

1. Adams, S. F., and Wilder, R. M. The surgical risk of the diabetic patient. Surg. Clin. N. Am., 1924, vii, 587–593.
2. Addis, T. The preparation of diabetic patients for operation. J. Am. M. Ass., 1915, lxiv, 1130–1134.
3. Allen, F. M., and Sherrill, J. W. Clinical observations with insulin; the use of insulin in diabetic treatment. J. Med. Research, 1922, ii, 803–985.
4. Allen, F. M. The timing of insulin doses. J. Am. M. Ass., 1924, lxxxii, 1937.
5. Banting, F. G., Campbell, W. R., and Fletcher, A. A. Insulin treatment of diabetes mellitus. J. Med. Research, 1922, ii, 547–604.
6. Berkman, D. M. Pre-operative and postoperative study of diabetic patients with surgical complications. Minnesota Med., 1921, iv, 28–40.
7. Binney. Quoted by Muller in Surg. Clin. N. Am., 1924, iv, 147–162.
8. Blum, L. Sur les indications des interventions chirurgicales dans le diabete sucre. Paris méd., 1919, xxxi, 341–343.
9. Brigham, F. G. Discussion of paper by Jones et al. Loc. cit.
10. Bruce, H. A. Surgical limitations in diabetes. Canadian J. Med. & Surg., 1914, xxxvi, 12–23.
11. Cantani, A. Ueber Diabetes mellitus. Deutsche med. Wchnschr., 1889, xv, 225, 252, 276.
12. Chantraine, H. Blutzuckeruntersuchungen bei Narkose und Nervenkrankheiten. Zentralbl. f. inn. Med., 1920, xxx, 521.
13. Chavannez, G. Contribution a l'étude de prognostic operatoire chez les glycosuriques. Gaz. heb. d. Soc. méd., 1903, xxiv, 116–120.
14. Cheever, D. Discussion of paper by Jones et al. Loc. cit.
15. Costain, T. E. Anæsthesia in diabetes. J. Am. Inst. Homeop., 1911, iii, 1078–1081.

16. CRUIKSHANK, W. Quoted by Murphy, Long Is. M. J., 1921, xv, 224–228.
17. CUMSTON, C. G. Diabetes and surgical operations. Boston M. & S. J., 1924, clxx, 316–317.
18. DELBET, P. Les interventions chirurgicals a le regime chez les diabetiques. Paris chir., 1921, xiii, 399–401.
19. DESANTELLE, W. T. Insulin in the treatment of diabetic complications. J. Am. M. Ass., 1924, lxxxii, 1471.
20. DEWES, H. Ueber Blutzuckeruntersuchungen bei Operationen in Lokal-anaesthesia und Athernarkose. Arch. f. klin. Chir., 1922, cxxii, 178–188.
21. DIEZ, S. Diabetes and trauma. Policlin., 1921, xxviii, 431–436.
22. EBSTEIN. Quoted by Kausch. Loc. cit.
23. FARR, R. E. Quoted by Berkman, Minnesota Med., 1921, iv, 28–40.
24. FISHER, D., and SNELL, M. W. The insulin treatment of pre-operative and postoperative non-diabetic acidosis. J. Am. M. Ass., 1924, lxxxii, 699–700.
25. FITZ, R. Surgical anæsthetics in diabetes mellitus. Med. Clin. North America, 1920, iii, 1107–1125.
26. FOSTER, N. B. A consideration of the surgical hazards in diabetic patients. Ann. Surg., 1920, lxxi, 382–386.
27. Idem. The treatment of severe diabetes with insulin. New York M. J. & Med. Rec., 1923, cxvii, 591–594.
28. FRERICHS. Ueber den Diabetes. Berlin: 1884.
29. GARDNER, G. W. Surgery in diabetes. Rhode Island M. J., 1922, v, 177–179.
30. GINSBERG, G. The use of insulin in a case of acidosis following trauma. J. Am. M. Ass., 1924, lxxxii, 1517.
31. GRIESINGER, W. Studien ueber Diabetes. Arch. f. phys. Heilk., 1859, iii, 1.
32. HARROP, G. A., JR. Management of Diabetes, Treatment by Dietary Regulation and the Use of Insulin. New York: Paul Hoeber, 1924, p. 60.
33. HIGGINS, F. A., and OGDEN, J. B. Traumatic glycosuria, observations made in 212 cases of head injuries. Boston M. & S. J., 1895, cxxxii, 197–204.
34. HIGGINSON. Sapræmic glycosuria. Brit. M. J., 1921, i, 296.
35. JONES, D. F., MCKITTRICK, L. S., and SISCO, D. L. Surgical treatment of gall-bladder disease in diabetics. Boston M. & S. J., 1924, cxci, 709–716.
36. JONES, A. T. Diabetes mellitus complicating surgery. Boston M. & S. J., 1923, clxxxviii, 483–488.
37. JOSLIN, E. P. The Treatment of Diabetes Mellitus. 3d ed., Philadelphia & New York: Lea & Febiger, 1923.
38. KAHN, M. Pre-operative preparation of diabetic patients and their subsequent treatment. Surg., Gynec. & Obst., 1920, xxxi, 363–365.
39. KAREWSKI, F. Diabetes und chirurgische Erkrankung. Deutsche med. Wchnschr., 1914, xl, 8–13.
40. KAUSCH, W. Trauma und Diabetes mellitus und Glykosurie. Ztschr. f. klin. Med., 1904, lv, 413–452.
41. KOERTE. Quoted by Bruce in Canadian J. M. & Surg., 1914, xxxvi, 12–23.
42. KONJETZNY, G. E., and WEILAND, W. Glykosurie und Diabetes bei chirurgischen Erkrankungen; Frakturen und Glykosurie. Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1915, xxviii, 860–891.
43. KRECKE, A. Diabetes und Chirurgie. Muenchen. med. Wchnschr., 1921, lxviii, 887–888.
44. KUELZ, E. Quoted by Kausch. Loc. cit.
45. LABBE, M. La chirurgie chez les diabetique. Ann. d. méd., 1918, v, 428–434.
46. LEYTON, O. Anæsthetics and diabetes mellitus. Lancet, 1920, i, 736.
47. MANGES. Quoted by Seelig in diabetes surgery. Am. J. M. Sc., 1924, clxviii, 495–502.
48. MULLER, G. P. Surgery in diabetics. Surg. Clin. North America, 1924, iv, 147–162.
49. MURPHY, J. P. Surgical aspect of diabetes. Long Island M. J., 1921, xv, 224–228.
50. PETTY, O. H. The practical use of insulin in the treatment of diabetes. Internat. Clin., 1923, ii, 36–54.
51. PHILLIPS, L. C. P. Some of the surgical aspects of glycosuria and diabetes. Lancet, 1902, i, 1308, 1386.
52. PLICQUE, A. F. La chirurgie chez les diabetique. Bull. méd., 1919, xxxiii, 482.
53. REISMAN. In Forcheimer, Therapeutics, vol. ii, p. 756.
54. SCHWARTZ. Quoted by Bruce in Canadian J. Med. & Surg., 1914, xxxvi, 12–23.
55. SEEGEN. Der Diabetes Mellitus. 3d ed. Berlin: 1893.
56. SMITH and DURHAM. Guys' Hosp. Reports, 1892, xlix, 335.
57. SPEESE, J. Insulin in surgery. Ann. Surg., 1924, lxxx, 146–147.
58. STETTON, D. The conservative treatment of diabetic gangrene of the lower extremity. J. Am. M. Ass., 1913, lx, 1126–1133.
59. STROUSE. Surg. Clin., Chicago, 1916, xxxvii, 37.
60. STROUSE, S., and SCHULTZ, O. T. Insulin in diabetes complicated by infection. J. Am. M. Ass., 1923, lxxx, 1592–1596.
61. THALHIMER, W. Insulin in treatment of postoperative (non-diabetic) acidosis. J. Am. M. Ass., 1923, lxxxi, 383; Insulin in treatment of toxemic vomiting of pregnancy. J. Am. M. Ass., 1924, lxxxii, 696–699.
62. TYTGAT, E. Le traitment des affections chirurgicales dans le diabete. J. de méd. de Par., 1916, xxxv, 40.
63. UMBER. Die Indikation und Prophylaxe chirurgischer Eingriffe bei Diabetikern. Deutsche med. Wchnschr., 1912, xxxviii, 1401–1403.
64. VERNEUIL. Bull. d. l. Acad. d. méd., Paris, 1881, x, 1461.
65. VON NOORDEN, WOODYATT, ALLEN, BOCK, and FITZ. Quoted by Joslin, loc. cit.
66. WEEDEN, W. M. The mortality of surgical complications in diabetes. J. Am. M. Ass., 1924, lxxxii, 1165–1166.
67. YOUNG, E. L., JR. Surgery and diabetes. Boston M. & S. J., 1923, clxxxviii, 767–769.

Since this paper was submitted for publication the following commendable articles have appeared:

CARRASCO-FORMIGUERA, R. Diabetes, surgical infections, and insulin. Lancet, 1925, i, 1576–1578.
FOSTER, N. B. Some surgical aspects of diabetes. J. Am. M. Ass., 1925, lxxxiv, 572–576.
GAGER, L. T. Surgical complications of diabetes under insulin treatment. Surg., Gynec. & Obst., 1925, lx, 630–635.

BENIGN TUMORS OF THE STOMACH

By E. L. ELIASON, M.D., F.A.C.S., and V. W. MURRAY WRIGHT, M.D., Philadelphia

By benign tumors of the stomach is meant adenomata, papillomata, myomata, fibromata, cysts, angiomata, lipomata, osteomata, myxomata and the misnomer "polyp." Under this head, diverticula, lymph node enlargements, hypertrophies, abscesses, aneurysms, and indurated swellings are not included.

Benign tumors of the stomach have been looked upon as "rare conditions." In comparison to their malignant fellows they are quite occasional, but an exhaustive survey shows that the reported cases are well on their way to the thousand mark.

Like any condition that is new or unusual, the subject of benign gastric tumors is one that has been attended with considerable confusion as regards diagnosis, classification, etc. It is the purpose of this article to bring before the profession at large a condensation of reported and collected cases together with a personal collection so that an opportunity is afforded for comparing cases, abstracting diagnostic features, and noting outstanding features in various types and so that a reference list is provided giving the special features. It is our hope that our readers will note that the condition is not so infrequent as it seems and that they will be able to give more thought to the unusual and undiagnosed cases; that they will make further use of the X-ray in gastric cases and operate (early) upon gastric cases that are benign or "doubtful" thus saving lives that would otherwise be lost.

ETIOLOGY

The etiology of the benign tumors of the stomach as a whole does not appear to be any different from that of the same tumors elsewhere in the body. No new features in connection with the following cases have been found. Among the causes given by various authorities are: chronic gastritis, alcoholism, dietetic errors, improper mastication and ætheroma of blood vessels. It is doubtful if any one of these causes is the specific agent any more than it would be for the same type of tumor elsewhere. Attention is called to the fact that in the majority of the personal cases of pedunculated "polyps" (many of which are myomata, adenomata and fibromata) chronic gastritis microscopically was proved a concomitant feature, that the hypertrophic form predominated, and that 80 to 90 per cent of the cases occurred among the laboring class. For the pedunculated types of tumor, especially those located in the pyloric end, it is the authors' belief that a low grade inflammation of the mucosa occurs, as a result of chronic irritation (either physical, nutritional, functional, chemical or bacterial) causing a local hypertrophy (see Case 39). Once the hypertrophy forms, it is increased mechanically by contractions of the stomach, peristaltic waves, and the pressure of the gastric contents as they are forced by on their way to the pylorus. This results in their being "pushed along" or lengthened out in the general direction of the pylorus. As the tissue continues to grow it does so at the expense of the stalk or pedicle which is stretched by the engulfing food being forced past and around it. Later when it is sufficiently large and long enough to do so, it is swept or carried into the pylorus by a peristaltic wave and produces the typical "ball-valve-syndrome" attack of pain.

A case recently operated upon by the senior author illustrates this "pulled" condition of the gastric mucosa. The patient was a male, aged 56, with a clinical and X-ray diagnosis of benign tumor of the stomach, prolapsing through the pylorus. At operation a doughy mass was felt projecting through a dilated pylorus into the duodenum. During palpation this mass slipped back into the stomach and could not again be pushed through into the duodenum. Gastrotomy disclosed the fact that the gastric mucosa around the entire circumference of the prepyloric area was very freely movable on the muscle coat and could be pushed along by the finger through the pylorus, causing an intussusception of a complete gastric mucous membrane cuff.

Basch notes that all polypoid cases are accompanied by chronic gastritis. Hauser and

others, cited by Bryan, believe gastric polyposis to be due to chronic inflammation. Konjetzny has demonstrated the progression of gastritis to adenoma and thence on to adenocarcinoma. Otto, cited by Bryan reported an adenoma in which was found a splinter of wood that had been swallowed. Zabel, according to Bryan, found large numbers of megastoma intestinale in the tufts of a large gastric papilloma with malignant degeneration. It is not at all impossible that low grade bacterial infection of the gastric mucosa due to a previous weakening or nutritional disturbance of the mucosa, may cause, in early stages, some of the hypertrophic conditions, which later develop into any of the various tumors.

SYMPTOMATOLOGY

Physical. In the majority of cases there is no pathognomonic sign. There are, however, a few "syndromes" which occur with certain types, viz. (1) gastric polyposis, (2) angiomata, (3) large myomata, (4) ball valve tumors. As the size varies in the different types from a split pea to that of a man's head, it will be readily seen that an entire absence of symptoms can occur and it may be possible definitely to palpate the tumor itself. The vast majority are less than a hen's egg in size. Many are internal, that is within the gastric cavity, some are intramural and a few are external or free in the abdominal cavity. Unless large, pedunculated, and external, they are not discoverable by physical examination. They are sometimes associated with gastric ulcers and carcinoma and any symptoms which they cause are lost in those of the associated condition.

The "syndromes" referred to above in the four mentioned types are:

1. *Polyposis.* In the presence of polyposis, we have indefinite gastric disturbances over a long period of time, gradually growing more and more severe, with loss of weight incommensurate with their appearance and symptoms, which are neither typical of gastric ulcer, carcinoma, or the usual gastric disturbances. A fairly typical picture is indefinite pains, becoming marked for 2 to 3 months before patient consults a physician, with no reference to meals, position, etc., except that the pain is relieved by frequent amounts of food taken in small quantities. At this stage the patients begin to lose weight rapidly and they become very weak and anæmic, with or without hæmatemesis or melæna and they present pictures that remind one of ulcer or malignancy, but the course is too rapid.

2. *Angiomata.* Depending upon the size, a variety of symptoms occur in the presence of angiomata. The symptoms vary from those of an acute gastritis to hæmorrhagic ulceration: hæmatemesis and melæna with spasmodic rapid loss of weight accompanying them; pain; then a temporary recovery, followed by a repetition of these symptoms, a marked anæmia, loss of weight, and occasionally an intermittent febrile condition. Ulceration no doubt is the cause of hæmorrhage when it occurs. If the growth is small, it may give no definite symptoms at all.

3. *Large myomata.* These growths may or may not have a pedicle. Because of their size they are sometimes palpable. The patient describes them as heavy lumps which change position and at times disappear—phantom tumors or external pedunculated myomata. When large, internal myomata may occlude or partially obstruct the passage of food and cause spasmodic contraction pains.

4. *Ball-valve tumors.* The "ball-valve-syndrome" is typical only of pedunculated internal tumors of the stomach, situated near the pylorus or in the pyloric half, and is characterized by repeated, paroxysmal, spasmodic attacks of gastric pain accompanied by temporary prostration, anorexia, anæmia, hæmatemesis or melæna, and rapid loss of weight. This is due to the temporary enveloping of the tumor and its pedicle by the pylorus. Later when it is freed, the patient's symptoms subside, he rapidly improves and continues in good health until another attack occurs, caused by the ball-valve action of the pedunculated tumor closing the pylorus. If relaxation does not take place or operation is not performed, intussusception of the pylorus and pyloric end of the stomach into the duodenum occurs with fatal results.

LABORATORY

Gastric analysis shows nothing typical. Achylia hypochlorhydria, normal acidity and

hyperacidity occur. Cases are reported with achylia and hyperacidity previous to operation which persisted after the removal of the tumors. The so-called egg-white-like mucus noted by Bryan and others was observed only once in the personal series of 50 reported. In rare cases where a gastric lavage has been performed, small pieces of tissue from the tumor have been recovered which when examined microscopically proved to be the same as that of the tumor subsequently removed at operation. Pieces of tissue from tumor fragments found in the stool may have become detached from intestinal tumors as pointed out by Struthers in his series of gastro-intestinal benign tumors. Hæmatemesis, melæna, low hæmoglobin and low red blood cell content occur in those cases in which ulcerations accompany the lesion or in which incarceration by invagination of the tumor occurs, as well as in cases with angioma.

ROENTGENOLOGY

By far the best diagnostic means at hand today in the diagnosis of benign tumors of the stomach, is the study of that organ by the X-ray. Though the picture is not commonly met with in X-ray work as some statistics would indicate, a scrutiny of the literature shows at least 50 cases that have come to X-ray. Once seen, it presents a picture which impresses one greatly as being so different from the picture of any other condition, that it may cause surprise and doubt when first observed. The photographic copies by Gassman, Balfour, Basch, Eusterman and Senty, Moore (a very good myoma), Matas; and a very clear ulcerating fibroma by Stetten are commended to the reader. Others who have noted the condition by X-ray and who give good descriptions are: Brin and Denecheau, Schlesinger, Heinz, H. Heinz, Konjetzny, Lederhose,McCullough, Neuber, Payr, Potherat, Dessecker, Myer, Stoner, Carman, Hahnes, Ruggles, Merrill, Paus, Geymueller, and Lieblein.

The single tumors which are large enough to be discernible stand out as globular, smooth, regular, clear, and persistent shadows, either on the lesser or greater curvature, and by their very smooth, uniform outline immediately strike one as not characteristic of the irregularity of malignancy and the scooped or "punched-out" areas of ulceration. Occasionally, as in the case of Neuber, Gassman, Brin and Denecheau, Basch, Eusterman and Senty, the pedicle itself is discerned as the barium trickles by it. It should also be noted that pedunculated tumors attached to the anterior or posterior walls have been overlooked at one X-ray examination to be found at a subsequent one when the patient was photographed in the "prone" position or was manipulated under the fluoroscope. The "prone" position best brings the tumor out in cases in which the tumor is not otherwise in apposition with the walls.

Hæmangiomata and cysts give a clear picture unlike the appearance of any other tumor and are quite typical. Moore gives the best pictures and reproductions.

Gastric polyposis gives a distinct mottled appearance not dissimilar to a bunch of grapes and is most frequently seen in the pyloric end. It reminds one of a "sponge" with its many punched-out mottlings.

Diagnosis by the position the tumor occupies in the stomach, cannot as yet be made with certainty according to types, though the attached table will show to the reader the commonest situations.

Schlesinger says benign tumors can only be diagnosed as such when the contour shows smooth round lines. Neuber points out a "bilocular appearance" in pedunculated myomata. Carman has seen very few cases (2) in 50,000 X-ray examinations of the stomach and considers the condition as relatively rare. Moore reviews 23 X-rayed cases and states: ". . . it seems that benign gastric tumors manifest certain signs roentgenologically which differ from those found in malignant or inflammatory lesions. If these signs are not characteristic, they are at least suggestive.

"1. They produce a filling defect that is circumscribed and punched-out in appearance.

"2. The filling defect is usually on the gastric walls leaving the curvature regular and pliant.

"3. While the rugæ are obliterated in the immediate area of the tumor, just as in inflammatory and malignant lesions: the rugæ

surrounding a benign tumor are more clearly normal in their arrangement and distribution.

"4. They cause little or no disturbance of peristalsis and retention is uncommon except when the lesion is at or very near the pylorus.

"5. They do not reveal a niche nor is there an incisura or other evidence of spasm.

"6. They are rarely sufficiently large to be palpated.

"Probably the most essential feature in the examination is the close and complete approximation of the walls of the barium filled stomach. This can be accomplished only by deep and thorough palpation and manipulation; thorough relaxation of the abdominal muscles is of course necessary. The patient should be rotated in both lateral directions and the stomach carefully scrutinized in the horizontal and vertical position. The solution of barium should be closely observed as it enters the cardia and passes over the posterior wall for occasionally a tumor projecting from the posterior wall will cause a splitting of the column, thereby giving the first appearance of its presence. A very small benign tumor is difficult and sometimes impossible to visualize; however, even a very small tumor near the pylorus will usually produce a definite filling defect.

"Differentiation roentgenologically of benign tumors and other gastric lesions can seldom be absolute, but in many instances the roentgenological signs warrant an attempt at such a distinction.

"Certain findings are strongly suggestive of their presence and when such signs are noted, the roentgenologist should hesitate to report the lesion as malignant and inoperable, especially if the clinical manifestations are indefinite."

PATHOLOGY AND TYPES

This description is a composite one of tumors collected to date and is quite general. The main features only are given as the detailed minutiæ vary so greatly.

Myomata. This type seems by far to outnumber all the others, averaging in our collected series nearly 60 per cent, this without including a number of the Deaver and Ashhurst collection. Fibromyoma, fibroleiomyoma, adenomyoma, leiomyoma, myoma, and adenoleiomyoma are included under this heading.

As a whole the myomata are hard, smooth, round or lobulated and circumscribed tumors, sessile or pedicled and lying free in the stomach, in the gastric wall or attached to the serous surface. The great majority of them seem to be serous or subserous. Their size ranges from that of a pea to a mass weighing 6,000 grams and unlike some of the other types which seem to have a predilection for the pyloric end, affect chiefly the anterior and posterior walls, lesser and greater curvatures.

The age at which they are most often found averages between 40 and 50; two cases as young as 20 and as old as 85 are reported. Females are more often affected than males.

They may be single or multiple but incline to the former. Ulceration, degeneration and malignant changes have been noted. Patterson notes that in 14 recorded cases secondary deposits "leio-myoma malignum" were found, while Basch (in Tice) has observed that histologically they are made up of unstriated muscle fibers, mixed with strands of fibrous tissue; that the submucous types are apt to undergo cystic degeneration, whereas the subserous or serous type are more prone to develop sarcomatous changes.

The review of a large number (310) of cases causes one to formulate the opinion that they are far from being as benign in their end-results as their benign classification suggests.

Papilloma. Under this heading are included the papilloma, adenopapilloma and the condition spoken of as papillomatosis. The latter term is reserved for cases which show a rather profuse collection or scattering of papillomata throughout the stomach and should not be applied to gastric cases which simply show a few multiple papillomata. Histologically, Delafield and Pruden observe: "In some cases of chronic gastritis there are small, polypoid hypertrophies (polypi) of the mucous membrane. Besides these we find polypoid tumors which may reach considerable size. They are composed of a connective-tissue stroma arranged in tufts covered with cylindrical epithelium, so that the tumor has partly the structure of an adenoma (papillary adenoma)."

Papilloma of the stomach is frequently classified by surgeons under the term "polypi." This should be discouraged as the two are distinct entities and the former are subject to ulceration and malignant changes.

Next to myoma, papilloma seems to be (7.8 per cent) the most common benign gastric tumor and were many of the so-called "polypi" examined histologically they would no doubt add materially to the number of recorded papillomata. Adenomatous characteristics sometimes change the microscopic appearance of the tumor to that of an adenopapilloma. This, likewise, should change our opinion of them as benign tumors since we are aware of the possibility of adenomatous structures undergoing malignant degeneration.

Papilloma seems to be on an average smaller than the myoma varying from a pea to a pear in size. The pyloric end of the stomach is more often involved than other areas. They may be pedicled as well as sessile. They are often multiple though only a few cases of papillomatosis are recorded. Males and females seem about equally affected. Forty to 50 years of age is the average age for their appearance, though they have been noted at the extremes of 20 and 71 years of age. Malignant changes are observed fairly often and McCallum has remarked that those which he has seen in the stomach "were associated with other tumors of a cancerous nature but that this was perhaps a coincidence and that they were of so soft and fragile a nature that losses of substance frequently occurred with hæmorrhage from the remaining surface."

Polypi. "Polypi" are poorly named pedunculated tumors. The term is indicative rather of a gross physical characteristic than any histological morphology. Microscopical examination shows that every so-called "polyp" belongs to one of the many known pathological tumors. In the tabulated series many benign tumors have been included under this heading because they were diagnosed grossly as such and no histological examinations were obtainable. In view of the fact that many of the gastric tumors which have been carelessly designated as polypi by pathologists and surgeons and which later have been shown, histologically, to be papillomata and adenomata (both of which are prone to malignant changes) an earnest plea is put forth for the discontinuance of this mis-leading and erroneous term.

Adenoma. They are benign tumors consisting of a central core of connective tissue, with tortuous, irregularly dilated tubular glands, which are lined with cylindrical epithelium and well supplied by blood vessels and lymphatics. Over these is spread a thin layer of unstriated muscle tissue which in turn is overlaid by a greatly hypertrophied mucous membrane.

McCallum notes that the glands are partly cystic and longer than usual, that they are embedded in a loose stroma and that because of traumatism, they are constantly inflamed. When fibrous tissue predominates they are termed "fibro-adenomata."

Grossly, the adenomata are round or lobulated projections from the interior of the stomach, and may be single or multiple, sessile or pedunculated. Cystic and carcinomatous degeneration occurs quite frequently.

In size they vary from a pea to a fetal head. Both sexes seem about equally affected. They are quite firm and frequently pedunculated. The lymphadenomata often have a creamy white color. The average age at which they occur is between 40 and 50 years. Adenomata, however, have been found in patients as young as 27 and as old as 76. Anatomically, the pyloric end of the stomach and the lesser curvature are most often involved. Ulcerations occur and malignant changes seem to be recently noted more than formerly as a result of the more frequent microscopic examinations of all tumors. In proportion to other benign tumors they rank about 5 to 6 per cent.

Cysts. Seven varieties are commonly described. Of the various 31 that have been collected and that represent 5 to 6 per cent of the benign tumors, 9 were gaseous, 5 were hydatid, 5 were hæmorrhagic, 3 were dermoids, 2 were traumatic, 2 were degenerative, 1 was embryonic (?) and 4 were not classified. Degenerative cysts should, when the type of tissue from which they are formed is known, be classified as that tumor, with cystic degeneration as a change or complication, i.e. fibroma, with cystic degeneration, etc.

The recorded cases vary in size from that of a nut to tumors weighing 1,000 grams. Most often they are serous or subserous. Males are perhaps more subject to them than females. They may occur at any age, depending on the nature of the cyst. A very young child has been reported and also a 73 year old man with a degenerative cyst.

A fluctuating palpable epigastric tumor steadily increasing in size following a history of an abdominal contusion should be regarded as a traumatic or hæmorrhagic cyst. Retention cysts have been observed in cases of chronic gastritis due to obstruction of the gastric gland ducts.

Lipoma. Microscopically the lipomata resemble those found in other parts of the body but sometimes they possess a few glands and muscle fibers when found in the submucous layer. They are lobulated and firm and, though most often interstitial, one has been noted with a pedicle. From the records it would be impossible to state what relation they bear to sex and age. They are much smaller than the myomata and usually vary between a hazel nut and a walnut in size.

The subserous variety seems twice as common as the submucous and according to Basch they are "usually solitary, situated mostly in the central part on the anterior wall, and rarely undergo cystic or malignant degeneration."

Fibroma. They may be single or multiple, sessile or pediculated, and are firm, smooth, and globular or elongated. They constitute about 5 per cent of the benign tumors and consist in the main of a fibrous tissue structure covered with mucous membrane. Their size varies from that of a pea to one that measured 12 by 6 by 4 centimeters. One-third of the fibromata have pedicles; the pyloric end is affected two to three times as often as the other areas, 60 per cent occurred in males, 50 years of age seemed the average and 16 and 71 were the extremes noted. Perhaps their relative frequency of situation in the pyloric end, is the reason they appear to simulate ulcer symptoms more than do the other types of benign tumors, and also why ulceration of their surfaces occasionally occurs.

"*Polyposis.*" This condition like that of the so-called "polyp" is a misnomer. Menetrier described it as *poly-adenome en nappe.* Once considered as quite rare it has now been found to be as common as the fibromata and more common than angiomata. Seventeen cases have been collected. As distinguished from multiple adenomata or papillomata, the tumors are quite small, very profuse, often situated in a fairly compact or circumscribed area, and with a hypertrophy and hyperplasia simultaneously of all the gastric glands more or less. The condition seems quite prone to ulcerative and malignant changes and the prognosis is not very good. They should be classified according to their microscopic characteristics under the respective headings of adenomata, papilloma, etc., which they are, as "adenomatosis gastrica," "papillomatosis gastrica," "fibromatosis gastrica," etc.

Angioma. Angiomata are generally single, smooth, soft, fair-sized (nut to an orange) and bluish-black or reddish tumors, which are frequently submucous, situated toward the body of the stomach and chiefly found in the anterior or posterior wall. A feeling similar to that of finding a mass of worms has been commented on as has also the fact that they are occasionally associated with similar tumors throughout the gastro-intestinal canal. They occur at varying ages and do not, as yet, seem to be confined greatly to any one period. They

TABLE I.—FREQUENCY OF DIFFERENT TYPES OF BENIGN GASTRIC TUMORS

Tumor	Collected series Number	Collected series Per cent	*Personal series Number	*Personal series Per cent
Myoma	321	57.3	4	8
Papilloma	44	7.8	16	32
"Polyps"	33	5.8	16	32
Adenoma	31	5.5	5	10
Cysts	29	5.17	1	2
Lipoma	28	5.	1	2
Fibroma	23	4.12	6	12
Lymphadenoma	14	2.5		
†Polyposis	11	1.9	1	2
Angioma	10	1.7		
Myxoma	3	0.5		
Osteoma	1	0.17		
Osteo-chondroma	1	0.17		
Unclassified	11	1.9		
	560		50	

*The above records (re 50 cases) embody 8,000 autopsies which were performed during 30 years at the I hiladelphia General and the University of Pennsylvania Hospitals, and do not include any collected cases from the literature. They also include 4 cases operated upon by the senior author.

†Besides the above 11 there were 5 adenomatosis, 2 papillomatosis and 1 fibromatosis, which were included under their respective headings.

TABLE II.—AUTHORS' CASES

Case	Sex	Age	Lesion	Location size and weight	Clinical features
1	M	58	*Adenomata	Scattered 1–10 m.m. (many)	Gastric symptoms. Also carcinoma of œsophagus at autopsy.
2	M	65	*Adenomata	Not stated Not stated	Discovered at autopsy. Bronchopneumonia.
3	M	65	*Adenomata (pedicle)	Pyloric end 1 cm. diameter	Chronic enteritis. Autopsy finding.
4	M	76	*Adenoma	Pyloric end Small	Pulmonary œdema. Autopsy. Microscopic adenoma and chronic gastritis.
5	M	50	*Adenomata	Lesser curvature and posterior wall Very small (very many)	Gastric symptoms indicated. Diagnosis: carcinoma of œsophagus. Autopsy: micro. adenoma, chronic gastritis with beginning malignancy.
6	M	52	*Lipoma	Submucous fundus	Lobar pneumonia. Autopsy.
7	F	85	*Myomata (subserous)	Serous surface 5 mm. (many)	Died of chronic myocarditis. Autopsy: several myomata of intestines also fibroma molluscum of body.
8	F	50	*Fibromyoma	Pyloric end Pea	Cerebral hæmorrhage. Autopsy.
9	F	62	*Fibroleiomyoma	Lesser curvature Pea	Chronic interstitial nephritis. Autopsy.
10	M	51	Fibroleiomyoma	Greater curvature 8 mm.	Lobar pneumonia. Autopsy.
11	M	71	*Fibromata (calcified)	Greater curvature Several	Operation for cholecystitis. Pulmonary œdema. Died. Autopsy findings.
12	F	60	*Fibromata	Fundus Pea (several)	Lobar pneumonia. Autopsy.
13	M	49	*Fibromata	Pyloric end Small (2)	Pulmonary tuberculosis. Autopsy.
14	M	65	Fibroma	Fundus Small	Chronic pleurisy. Emphysema. Autopsy.
15	M	58	*Fibromata (pedicle)	Pyloric end. Several pea-sized; 1, 4 by 12 cm.	Gastric symptoms 6 yrs. similar to malignancy. X-ray at 2 hospitals in 3 yrs. "Malignancy." Died. Autopsy: "fibromata, acute catarrhal gastritis, no carcinoma."
16	M	73	*Cyst (fibromatous)	Not stated Nut	Died of pneumonia. Autopsy.
17	M	45?	Cyst (hæmorrhagic)	Pyloric end 8 mm.	At autopsy.
18	F	68	*Papilloma	Near œsophagus 1 cm.	Cerebral hæmorrhage. Autopsy.
19	M	52	*Papillomata	Pyloric end Not stated (many)	Cirrhosis of liver. Ulcerative enteritis. Autopsy.
20	F	67	Papilloma (pedicle)	Pyloric end 18 mm.	Cholelithiasis. Aortic regurgitation. Autopsy.
21	M	49	Papilloma	Pyloric end 8 mm.	Organic dementia. Autopsy.
22	F	69	Papillomata	3—greater curv. 1—pyloric end 3—small 1–1.6 cm.	Cerebral hæmorrhage. Autopsy.

*History obtainable from autopsy records.

TABLE II.—AUTHORS' CASES—Continued

Case	Sex	Age	Lesion	Location, size and weight	Clinical features
23	M	71	Papillomata	Scattered 8–10 mm. (profuse)	Bronchopneumonia. Autopsy.
24	F	46	Papillomata	Pyloric end Small (2)	Cardiac dilatation. Purpura hæmorrhagica. Autopsy.
25	M	46	Papilloma	Pyloric end Grape	Ruptured dissecting aneurysm. Autopsy.
26	M	65	*Papilloma	Pyloric end Small	Autopsy. Histologic examination showed chronic hypertrophic gastritis also.
27	M	65	*Papilloma with pedicle	Middle of greater curvature 1 cm.	Aortic regurgitation, hydrothorax; autopsy; histological examination: papilloma and chr. gastritis.
28	F	65	Papilloma with pedicle	Pyloric end 8 mm.	Syphilis of brain and cord. Autopsy.
29	M	45	†Papilloma (pedicle)	Pyloric end	Symptoms of ulcer. X-ray at 2 hospitals. Diagnosis: carcinoma, cholelithiasis and renal calculi. Operation. Histological examination: papilloma with adenocarcinoma.
30	M	45	*Papillomata	Posterior wall near greater curvature Cherry	Symptoms: X-ray: papilloma of stomach. Operation. Papilloma of stomach. Histological examination: papilloma of stomach.
31	M	44	†Papilloma (pedicle)	Pyloric end 3 by 2 cm. (ribbon-like)	Pain in epigastrium 3 mos. X-ray "retention." Diagnosis: duodenal ulcer. Operation. Papilloma pyloric end; carcinoma of cardia; histological examination: papilloma with carcinoma.
32	F	26	†Papilloma (pedicle)	Anterior wall 1.5 cm. long	Duodenal ulcer symptoms. Operation. Gastric ulcer and papilloma. Autopsy. Histological examination: same.
33	M	25	†Papillomatosis	Diffuse Pea-grape	Symptoms: X-ray: Operation. Gastric ulcer, healed; duodenal ulcer; diffuse papillomata. Histological examination: papillomata and ulcer. "Polyposis."
34	M	40	Polyp (pedicle)	Pyloric end 15 mm.	Progressive muscular atrophy autopsy.
35	M	60	Polyposis	Pylorus and cardia Pea (diffuse)	Stricture of œsophagus, tertiary lues. X-ray (for œsophagus) condition not seen; gastrotomy for stricture; died; autopsy.
36	M	69	Polypi with pedicle	Pyloric end 1 cm. (3)	Bronchopneumonia; autopsy.
37	M	58	Polypi	Pyloric end 8 mm. (several)	Cardiac dilatation; autopsy.
38	F	49	Polypi	Pyloric end (2)	Hypostatic pneumonia. Autopsy.
39	M	60	Polypi	Pyloric end 5 to 7 mm. (sev.)	Lobar pneumonia. Autopsy.
40	M	49	Polyp	Œsophageal end 8 mm.	Delirium tremens. Autopsy.
41	M	77	Polypi	Pyloric end 5–8 mm. (2)	Empyema. Autopsy.
42	M	75	Polyp	Pyloric end	Pulmonary tuberculosis. Autopsy. Polyp almost closed pyloric orifice.

*History obtainable from autopsy records.
†Cases No. 29, 31, 32, and 33 were operated upon by the senior author.

TABLE II.—AUTHORS' CASES—Continued

Case	Sex	Age	Lesion	Location, size and weight	Clinical features
43	M	42	Polypi	Pyloric end 8 mm. (pea) (several)	Pulmonary tuberculosis. Autopsy.
44	M	56	Polyp	Pyloric end 6–12 mm.	Hepatic cirrhosis. Autopsy.
45	F	81	Polyp	Middle of greater curvature 3 cm.	Chronic myocarditis. Autopsy.
46	M	75	Polypi	Middle of stomach 2–3 cm.	Insane. Autopsy.
47	M	56	Polyp	Lesser curvature 2 cm. diameter	Gastric complaint for years. X-ray: carcinoma. Autopsy: polyp and carcinoma. Histological examination: carcinoma, polyp not sectioned.
48	F	81	Polyp	Middle of greater curvature 3.3 cm.	Chronic myocarditis. Autopsy.
49	M	65	Polyp	Not stated Small	Myocarditis, cellulitis of leg. Autopsy.
50	F	49	Polypi	Not stated (pyloric?) Marble (2)	Gastric symptoms for years pointing to malignancy. Autopsy: polyps but no carcinoma.

ulcerate easily and hæmorrhages are apt to occur. Two have been noted with a pedicle though they are most often interstitial. Lemon reports a case with sarcomatous changes.

Myxoma. Myxomata are generally gelatinous, encapsulated, and semi-transparent tumors situated in the walls of the stomach and covered by mucous membrane. They are uncommon and no doubt due to myxomatous degeneration of myxomata or other tumors.

Osteoma. Osteomata are exceedingly rare tumors. Wade reports a doubtful osteoma and Eerkles an osteochondroma of the gastric wall.

Lymphadenoma. According to Deaver and Ashhurst, Gilly collected in 1856 51 cases of gastro-intestinal lymphadenoma, the stomach being involved in 14 instances. A few cases have been recorded since. In all known cases lymphomatous growths have been observed in other parts of the body as well, in the spleen, lymph nodes, bones, pharynx, or intestines. In all cases of gastric lymphadenoma, the intestines were involved. This affection arises either in the subserous or submucous lymphatic tissue of the stomach. In the submucous tissues it exists either as a localized or diffused form, usually manifesting itself on the surface of the stomach by a polypoid condition of the mucosa. Ulceration is more usual in the circumscribed form. The tumors which arise in the submucous tissues rarely cause obstruction

TABLE III.—SUMMARY OF AUTHORS' 50 CASES

	Cases	Per cent
Polypi	16	32
Papillomata	16	32
Adenomata	5	10
Myomata	4	8
Fibromata	6	12
Cysts	2	2
Lipoma	1	1
Polyposis	1	2

The summary analysis of the authors' cases (Table III) seems at wide variance with that of the collected cases (Table I). We are sorry to state that 19 microscopical examinations only were found in the records of the 50 cases, 3 of the examinations concerned only the ulcer or malignancy accompanying the benign tumor, leaving 16 histological examinations. Of this number

	Per cent
5 were adenomata (5/16)	31.25
4 were papillomata	25
4 were fibromata	25
2 were myomata	12.5
1 was a cyst	6.25

Had the so-called "polypi" been histologically examined they would naturally have altered the percentage of tumor types and made an accurate classification more possible, probably showing adenomata and papillomata as the predominating types.

It must be remembered that comparatively few of the cases in the collected series were examined microscopically and that accurate classification of all tumors is possible only when the profession insists that every tumor removed be sectioned histologically and reported.

SUMMARY OF TABLE II

POLYPI—17 CASES

Location — Cases
- Pyloric end — 10
- Œsophageal end — 1
- Cardia — 1
- Middle — 1
- Not stated — 2
- Greater curvature — 1
- Lesser curvature — 1

Number
- Multiple — 9
- Single — 8
- Polyposis — 1

Size
- 5 mm. to 3.3 cm.
- Pedicled — 2

Sex
- Male — 13
- Female — 9

Age — Years
- Average — 61
- Youngest — 40
- Oldest — 81

Found — Cases
- At autopsy — 17

General symptoms
- Four complaints.

Complications
- Case 35, stricture of œsophagus.
- Case 47, carcinoma.

X-ray
- Used, Cases 35 and 47.

Remarks
- In Case 35 gastrotomy for stricture of œsophagus. Also X-rayed but condition not discovered until autopsy. In Case 42 tumor almost closed the pylorus. X-ray examination Case 47 made twice in 3–4 years but carcinoma only recognized. In Case 50 an incorrect diagnosis of carcinoma was made.

PAPILLOMATA—16 CASES

Location — Cases
- Pyloric end — 10
- Œsophageal end — 1
- Anterior wall — 1
- Posterior wall — 1
- Scattered — 2
- Greater curvature — 1

Number
- Multiple — 6
- Single — 10

Size
- 8 mm. 3 cm.
- Pedicled — 6

Sex
- Male — 10
- Female — 6

Age — Years
- Average — 50
- Youngest — 25
- Oldest — 71

Found — Cases
- At autopsy — 12
- At operation — 3
- By X-ray and operation — 1

Gastric symptoms
- Present in Cases 19, 20, 29, 30, 31, 32, 33 — 7

Complications
- Cholelithiasis and carcinoma, Case 29 — 1
- Cholelithiasis and aortic regurgitation, Case 20 — 1
- Ulcerative enteritis, cirrhosis of liver, Case 19 — 1
- Gastric ulcers, Cases 32 and 33 — 2
- Carcinoma of cardia, Case 31 — 1

X-ray
- Cases 29, 30, 31, 33 — 4
- Condition recognized Case 30 — 1

Remarks — Cases
- Histological examination showed
  - Chronic gastritis — 2
  - Ulcers — 2
  - Carcinoma — 2
- In Case 29 benignancy with malignancy not recognized in 2 X-ray examinations. In Case 30, X-ray, operative and pathological diagnoses all papilloma of stomach. In Case 33—polyposis gastrica.

ADENOMATA—5 CASES

Location — Cases
- Pyloric end — 2
- Scattered — 1
- Not stated — 1
- Lesser curvature and posterior wall — 1

Number
- Multiple — 2
- Single — 2
- Not stated — 1

Size
- 1–10 mm. to 1 cm.
- Pedicled — 1

Sex
- Male — 5
- Female — 0

Age — Years
- Average — 63
- Youngest — 50
- Oldest — 76

Found — Cases
- At autopsy — 5
- At operation — 0
- By X-ray — 0

Gastric symptoms
- Present, Cases 1, 3, 5 — 3

Complications
- Carcinoma of œsophagus Cases 1 and 5 — 2
- Chronic gastritis, Case 4 — 1
- Chronic enteritis, Case 3 — 1
- X-ray — 0

Remarks
- In Case 5—histological examination showed chronic gastritis with early malignancy.

MYOMATA—4 CASES

Location — Cases
- Pyloric end — 1
- Lesser curvature — 1
- Greater curvature — 1
- Serous surface — 1
- Subserous surface — 1

Number
- Multiple — 1
- Single — 3

Size — Cases
- 5 to 10 mm.
- Pedicled — 0

Sex
- Male — 1
- Female — 3

Age — Years
- Average — 49
- Youngest — 50
- Oldest — 85

Found — Cases
- At autopsy — 4
- At operation — 0
- By X-ray — 0
- General symptoms — 0

Complications
- Fibroma molluscum of body with myomata of uterus (Case 7) — 1
- X-ray — 0

Remarks
- Fibroleiomyoma — 2
- Myoma — 1
- Fibromyoma — 1
- Subserous myoma (Case 7) — 1

CYSTS—2 CASES

Location — Cases
- Pyloric end
- Not stated — 1

Number — Cases
- Multiple — 0
- Single — 2
- Pedicled — 0

Size
- 8 mm. to 2.5 cm.

Sex
- Male — 2
- Female — 0

Age — Years
- Average — 59
- Youngest — 45
- Oldest — 73

Found — Cases
- At autopsy — 2

Gastric symptoms
- Noted — 0

Complications
- Noted — 0
- X-rayed — 0

Remarks
- Fibromatous cyst — 1
- Hæmorrhagic cyst — 1

LIPOMATA—1 CASE

Location — Cases
- Submucous in fundus — 1

Number
- Single — 1

Sex — Cases
- Male — 1
- Female — 0
- Age, years — 52

Found at autopsy
- No gastric symptoms; died of lobar pneumonia.

Complications
- Pneumonia

FIBROMATA—5 CASES

Location — Cases
- Pyloric end — 2
- Fundus — 2
- Greater curvature — 1

Number
- Multiple — 4
- Single — 1

Size
- Pea to 4 by 12 cm.
- Pedicled — 1
- Sessile — 4

Sex
- Male — 4
- Female — 1

Age — Years
- Average — 60
- Youngest — 49
- Oldest — 71

Found — Cases
- At autopsy — 5
- At operation — 0
- X-ray — 0

Gastric symptoms — Cases
- Referable to gall bladder
- Case 11 — 1
- Case 12 — 1

Complications
- Cholecystitis (Case 11) — 1

Remarks
- Case 12 had gastric symptoms for 6 years, believed to be cancer. In 3 years X-rayed at 2 hospitals and diagnosis of cancer made. Autopsy showed fibromata. ac. Cat. Gast. No malignancy.

Summary of complications (abdominal).

Gastritis	3
Carcinoma of stomach	3
Ulcers	2
Cholelithiasis	2
Renal calculi	1
Enteritis	1
Cirrhosis of liver	1
Carcinoma of œsophagus	1
Stricture of œsophagus	1

Summary of ages	Cases	Per cent
20 to 30 years	2	4
30 to 40 years	..	0
40 to 50 years	12	24
50 to 60 years	10	20
60 to 70 years	16	32
70 to 80 years	7	14
80 to 90 years	3	6

These ages do not signify age of occurrence of tumors, but rather the age at which they were found; at autopsy in most cases, previously undiagnosed.

but those commencing in the subserous tissue which are usually diffuse, frequently penetrate, paralyze and ultimately destroy the muscular coat, producing dilatation of the stomach and consequent stagnation of food. In some cases it appears to have been demonstrated that the disease originated in the neighboring mesenteric lymph nodes, and subsequently involved the subserous lymphatic structures of the stomach.

BIBLIOGRAPHY

1. Amelung. Pedunculated myosarcoma of the gastric wall. Deutsche med. Wchnschr., 1913, xxxix, 973.
2. Balfour, D. C. Polyposis of the stomach. Surg., Gynec. & Obst., 1919, xxviii, 405–407.
3. Basch, Seymour. Primary benign growths of the stomach. Surg., Gynec. & Obst., 1916, xxii, 165–170.
4. Baylac and Dieulafe. Cited by Matas.
5. Berger. An unusual case of tumor of the stomach. Veroeffentl. a. d. Geb. d. Mil.-San.-Wes., Berl., 1906, xxxv, 330–342.
6. Berries, G. V. T. Myomata of the stomach. Hops.-Tid., Kobenh., 1918, lxi, 1617–1629.
7. Bland-Sutton, Sir J. On fibroids, lipomas, dermoids, and polypi of the stomach and intestines. Lancet, Lond., 1920, i, 5–9.
8. Boquet, H. Myoma of the stomach; partial gastrectomy. Arch. d. mal. de l'appar. digest. etc., Par., 1919, x, 425–427.
9. Brin and Denecheau. Fibroma of the stomach, removed by a large gastrectomy. Bull. et mém. Soc. de chir. de Par., 1922, xlviii, 148–150.
10. Brodowski. Cited by Berger.
11. Bryan, Lloyd. Gastric polyposis. Southwest. Med., Phoenix, Ariz., 1922, vi, 223–227.
12. Cantwell. Cited by Basch.
13. Cernezzi. Cited by Berger.
14. Cheyne, D. G. A case of fibromyoma of the stomach. Brit. M. J., 1912, i, 118.
15. Chosrojeff. Two cases of rare tumors of the stomach: (1) polyposis ventriculi; (2) endothelioma of the stomach. Beitr. z. path. Anat. u. z. allg. Path., Jena, 1912, liv, 595–613.
16. Deaver and Ashhurst. Surgery of the upper abdomen.
17. Doering. Cited by Bryan.
18. Du Bray, E. S. Gastric polyposis (papillomatosis) operation; presumable cure. Arch. Int. Med., 1920, xxvi, 221.
19. Eastman, J. R. Leiomyoma of the pylorus. New York M. J., 1916, civ, 785–88.
20. Eberle, A. Gastric pediculated fibroma with fibromata of the gastro-intestinal tract. Khirurg. Arkh. Velyaminova, St. Petersburg, 1913, xxix, 988–95.
21. Ebstein. Cited by Bryan.
22. Eerkes, L. E. A case of osteochondroma of the stomach wall. Nederl. Tijdschr. v. Geneesk., Amst., 1919, ii, 1769.
23. Eisenbery, A. B. Report of a case of fibromyoma of the stomach. Proc. New York Path. Soc., 1913, n.s., xiii, 35.
24. Ellis, A. G. Pedunculated fibroma of the stomach. Proc. Path. Soc., Phila., 1918, n.s., xxi, 1.
25. Erdman, J. F. Fibroma of the cardia in a girl of 18 with gastrotomy and enucleation. Am. J. Obst., N. Y., 1913, lxviii, 964–966.
26. Erloch. Cited by Basch.
27. Eusterman, G. B., and Senty, Elmer G. Benign tumors of the stomach. Surg., Gynec. & Obst., 1922, xxiv, 5–15.
28. Finney, J. M. T., and Friedenwald, Julius. Gastric polyposis. Am. J. M. Sc., 1917, cliv, 683.
29. Gassman, Fritz. Roentgenologic diagnosis of polypus of the stomach. Arch f. Verdauungskr., Berl., 1921, xxviii, 226–230.
30. Glenn, R. A. Ulcerating myoma of the stomach. Proc. Path. Soc., Phila., 1914, xvi, 9.
31. Heinz. Polyposis of the stomach. Cor.-Bl. f. schweiz. Aerzte, Basel, 1912, xlii, 354.
32. Heinz, H. Polyposis ventriculi. Beitr. z. klin. Chir., 1914, xciii, 228–250.
33. James, J. E., Jr., and Sappington, S. W. Myoma of the stomach; report of a case successfully removed. Surg., Gynec. & Obst., 1915, xxi, 744–746.
34. Konjetzny, G. E. Fibroma of the stomach. Beitr. z. klin. Chir., 1920, cxix, 53–61.
35. Lamb, D. S. Another case of polyposis gastrica (polyadenoma). Washington Med. Ann., 1914, xiii, 180.
36. Ledderhose, G. Polypi of the stomach. Deutsche med. Wchnschr., 1913, xxxix, 2349.
37. Leiblein. Fibroma of the stomach. Med. Klin., Berl., 1921, xvii, 1583.
38. Lemon, W. S. Angioma of the stomach. Med. Rec., N. Y., 1920, Feb.
39. Lotsch. Invagination of a myoma of the pylorus. Deutsche med. Wchnschr., 1912, xxxviii, 1164.
40. Magnus, Alsleben, and Cohen. Cited by Neuber.
41. Matas, Rudolph. Pediculated polypoid fibro-adenoma of the stomach of ball-valve type, causing an unusual and complex syndrome. Surg., Gynec. & Obst., 1923, xxxii, 723–731.
42. McArthur, L. L. Fibromyoma of stomach simulating gastric ulcer. Surg. Clin., 1918, ii, 1233–1238.
43. McCarthy, W. and Glomset, D. J. Fibroma of the stomach. J. Iowa St. M. Soc., 1915, v, 409–412.
44. McCarthy and Hektoen, L. Submucous fibroma of the stomach with perforation of the mucosa and pyloric obstruction. Tr. Chicago. Path. Soc., 1913–15, ix, 237–240.

45. McCULLOUGH, J. P. Gastric polypus and X-ray demonstration of the same. J. Radiol., Omaha, 1923, iv, 61–63.
46. McPHEDRAN, A. Multiple polypi in stomach (gastric polyposis). Canad. M. Ass. J., 1921, xi, 524.
47. MILLS, PERCIVAL G. Multiple polypi of the stomach (gastritis polyposa). Brit. M. J., 1922, x, 226–231.
48. MOORE, ALEXANDER B. A roentgenologic study of benign tumors of the stomach. Am. J. Roentgenol. & Radium Therap., 1924, xi, 61–66.
49. NEUBER, ERNST. Operation on a pedunculated myoma of the stomach. Wien. klin. Wchnschr., 1920, xxxiii, 838–840.
50. NOVAK, EMIL. Polypoid adenoma of the stomach. J. Am. M. Ass., 1920.
51. OBERNDORFER. A pendulous, cavernous lymphangioma of the external surface of the stomach. Beitr. z. path. Anat. u. z. allg. Path., Jena, 1921, lxix, 418–421.
52. ORTH. Cited by Basch.
53. OUTLAND, J. H., and CLENDENNING, L. Myoma of the stomach. Ann. Surg., 1913, lviii, 812–817.
54. PAYR. Specimen of an unusually large polypus of the stomach. Muenchen. med. Wchnschr., 1917, lxiv, 325.
55. PERL NEELSON. Cited by Basch.
56. PÓLYA, E. Myoma. Pest. med. chir. Presse, Budapest, 1912, xlviii, 221.
57. POTHERAT, E. and G. Large pedunculated tumor of the stomach; gastrotomy; ablation; partial gastrectomy. Bull. et mém. Soc. de. chir. de Par., 1913, xxxviii, 161–164.
58. RANKIN, W. Papilloma of the posterior wall of the stomach, removed by transgastric oper. Glasgow M. J., 1920, xciii, 185.
59. RUPPRECHT. Cited by Basch.
60. SHERREN. Cited by Basch.
61. STEINER, RUDOLF. Myomata of the gastro-intestinal tract. Beitr. z. klin. Chir., 1898, xxii, 1–112, 407–440.
62. STETTEN, D. The submucous lipoma of the gastro-intestinal tract. Surg., Gynec. & Obst., 1909, ix, 156–176.
63. Idem. Ulcerating fibroma of stomach simulating malignancy. Ann. Surg., 1920, May.
64. STRUTHERS, John E. Multiple polyposis of the gastro-intestinal tract. Surg., Gynec. & Obst., 1924, 610–624.
65. THOMAS, A., and GRAHAM, J. M. Fibromatosis of the stomach and its relationships to ulcer and to cancer. Tr. Am. Surg. Ass., 1913, xxxi, 13–46.
66. TILGER. Cited by Chosrojeff.
67. VERSE. Cited by Chosrojeff.
68. VIRCHOW. Cited by Berger.
69. VON GRIEDRICH, L. A case of myoma of the stomach. Deutsche med. Wchnschr., 1920, xlvi, 1139.
70. WILENSKY, A. O. Benign stomach tumors. Prog. Med., Phila., 1923, ii, 104.
71. Idem. Polypoid adenoma of the stomach. Prog. Med. Phila., 1921, ii, 110.

A CONSIDERATION OF THE CERVIX UTERI FROM THE STANDPOINT OF ITS SPHINCTER PROPERTIES

By E. MURRAY BLAIR, M.D., C.M., Vancouver, British Columbia

PROBABLY it is safe to say that the cervix is the commonest seat of chronic infection in the female anatomy. Few women, and, I believe, no parous women, go through life without some degree of infection stored away in those tortuous cervical glands. As would be expected in this age of hyperactivity concerning focal infections, much has been written from a clinical standpoint on cervical infections, acute and chronic, and their treatment. Numerous articles by the outstanding men of the profession and others show that much study has been applied to the clinical treatment of this organ, so prone to misfortune. The thing that strikes one most forcibly, I think, is the fact that these brilliant minds, working with one common aim in view—the eradication of infection in this area—advocate such widely varied methods. One emphasizes amputation, another radium, another suction and active hyperæmia, another cautery, and so on through a seemingly never ending list of methods.

It was recently our privilege to visit some 250 of the leading hospitals of northeastern America, and as I was particularly interested in gynecological and obstetrical problems, perhaps special attention was paid to those phases of the work. The impressions gained from my observations as to the treatment of chronic endocervicitis are here offered for the consideration of the profession in the hope that a lesson may be learned even from very chaos. These impressions are the result of visits not only to the smallest hospitals in the outlying districts but to the largest clinics in the medical centers of our land.

As in our studies, so in our observations, we are struck first of all by the very multiplicity of methods seen. Various local applications, tampons, operations of every description, cauteries, and occasionally radium are advocated. There seems to be an entire lack of unity of opinion in a given district, city, and sometimes even in the same clinic. Every surgeon we talked with was a law unto himself in the matter. He seemed quite satisfied with his own method of procedure and he quoted his results in substantiation.

Especially is one impressed, we think, with the enormous amount of surgery everywhere performed on the cervix and on patients of all ages for various reasons and by diverse methods. Trachelorrhaphies are apparently done oftener in the smaller towns than in the larger cities. Everywhere, however, amputation is rampant. The methods of amputation are many, ranging from the old Schroeder, with all its modifications, to the newer Sturmdorf method, with all the modifications which must naturally evolve therefrom. It was a distinct surprise though, to find a comparatively small number of surgeons throughout the country using the Sturmdorf amputation or any modification of it.

The electrocautery was then only in the process of introduction in the larger clinics. It was being advocated in those cases of chronic cervical infection without complications, i.e., with no indications for surgical interference elsewhere. Otherwise straight surgical procedures on the cervix were employed with exclusion of the cautery.

Radium, as a curative treatment for chronic endocervicitis was being used in only a few of the larger clinics.

Decidedly at sea, and in a seeming maze of treatments, we were privileged to discuss our perplexity with an eminent professor of gynecology and obstetrics in a well known University. His remarks will always be remembered, and they are really the basis of this study. He said: "Always keep in mind that the cervix uteri has all th e properties and duties of a sphincter and treat it with the same consideration and reverence you would a sphincter elsewhere in the body. You will then approach the cervix with the proper attitude." The statement is, to me, an inspiration, and though its anatomical and

physiological accuracy will undoubtedly be questioned by many, it provides, we believe, the proper attitude for the rank and file of our surgeons.

Chronic endocervicitis, if at all marked, demands treatment and, if possible, cure. Its presence may cause discomfort physically and mentally, may be a focus of infection causing trouble elsewhere, may cause sterility. As treatment, then, is a necessity in many cases, is there no hope that some degree of uniformity of treatment may be established? Why should there be a multiplicity of methods in vogue as there is at present? Many of these methods doubtless accomplish their purpose, but many not only fail but do infinite harm.

It is believed that the answer lies, to a great extent, in the fact that the cervix *as an organ*, has been neglected in the past by university teachers and those who guide the channels of medical thought. Too long has the cervix been considered as merely the "neck of the womb," a passageway of decidedly passive interest except during the first stage of labor or in the presence of leucorrhœa.

As far as can be ascertained, after diligent search with the aid of the library research department of the American College of Surgeons, every standard or recognized textbook in the language, whether anatomical, histological, physiological, pathological or clinical, is content to describe the uterus in detail and allow the student to consider the cervix as subservient and merely adjunct. The histologists apparently show the most respect, then the pathologists; the anatomists practically ignore it and the physiologists disregard it completely. Small wonder then for the lack of reverence.

It is our intention to consider the cervix as a separate organ which is so highly specialized as to border on the mysterious, and which performs a function indispensable to the continuation of the race. That function must surely be based on anatomical and physiological principles, and a knowledge of these principles is essential to understand the function of the cervix. A knowledge of cervical function is essential in the prescribing of treatment.

IS THE CERVIX A SPHINCTER

One is well aware that a number of authorities do not consider the cervix a sphincter. Probably this is the general trend of opinion today. This argument is based chiefly on the anatomical fact that there are no circular muscle fibers which completely encompass the cervix. Uterine muscle fibers sweep down, at least schematically, from an origin near the bases of the fallopian tubes and by a series of spirals partially encompass the cervix, but no single fiber completely surrounds it. We believe that the whole cervical musculature is probably thus made up. Therefore as every muscle fiber contracts toward its fixed point, the base of the fallopian tube, the cervix, as such, is devoid of function and "physiologically is nothing more than a passive communicating duct between the vagina and uterine cavity proper" (Sturmdorf).

This theory of cervical musculature has always appealed to me, as far as it goes. We visualize that form of a tobacco pouch which is made in the shape of a bag, with the sides pressed into spiral folds in such a way that a turn to the right causes the folds to fit together and the bag collapse shut; by an opposite turn the bag is opened and the contents can be removed.

We like its application to the first stage of labor. How nicely it explains the drawing up of the cervix, the obliteration of the internal os, the gradual thinning out of the lower uterine segment and the resulting dilatation. To apply the above, however, to the 6 months immediately before labor is not so easy. It is satisfactory as far as it goes, but it does not go far enough. The utterly passive nature of the cervix of the pregnant uterus is questioned; the cervix is, and should be so considered, an organ, capable of function and with a remarkable work to do.

PHYSIOLOGY

The uterus, like the heart, and indeed all hollow muscle organs of the body, is fundamentally myogenic in action; that is, the source of the action of the uterus is placed in the muscle itself. Its function, as in the heart, may be described as rhythmicity, excitability, contractility, conductivity, and tonicity.

In other words, the muscular fibers of the uterus possess the power of rhythmically creating a stimulus, of being able to receive a stimulus, of responding to a stimulus by contracting, of conveying the stimulus from muscle fiber to muscle fiber, and of maintaining a condition called tone.

Now, in the normally functioning pregnant uterus, there are two natural forces acting on the gestation: (1) the force of gravity, (2) intra-uterine pressure. The first force is constant and gradually increasing, the second is regularly intermittent, depending on the rhythmicity of the uterus as to time and in the other four functions as to length and severity. The force of gravity in the human is unfortunately directed against the one vulnerable point in the uterine armor—the cervix. The cervix of no other vertebrate has this force with which to contend. Is the cervix then an entirely passive organ subjected constantly to the two forces above described? Before arriving at a conclusion we must first deal with that little understood phenomenon of smooth muscle—*contraction, tone.*

Concerning the tonicity of voluntary or striated muscle, a good deal has been learned. We know that voluntary muscle will react to tonic contractions for a certain length of time and will finally become exhausted and cease to function. We know that voluntary muscle is wholly dependent on impulses from the central nervous system to preserve its tone or set it into activity.

On the other hand involuntary or smooth muscle possesses an automatic tone and activity manifested in rhythmic contraction and relaxation, entirely independent of the central nervous system. Smooth muscle will react to impulses from the central nervous system, it is true, but it is only reflex action, not voluntary control. Smooth muscle retains its tonicity indefinitely regardless of nervous impulse, voluntary muscle cannot. The tonicity of smooth muscle goes on indefinitely, we believe, without appreciable expenditure of energy or resulting exhaustion. This is known as the smooth muscle tone phenomenon or "unvarying minimal contraction" peculiar to smooth muscle. Probably nowhere in the body is smooth muscle tone phenomenon so well seen rising to meet an emergency as in the pregnant uterus. Its expression is the well known "Hegar's sign." It is one of the very earliest signs of pregnancy. Hegar's sign, though an apparent softening of the lower uterine segment is in truth a smooth muscle contraction with resultant neighboring lymphatic engorgement. It does not appear at any exact, invariable location. Like the internal os it is without anatomical or histological definition but with a definite physiological alignment.

Here is nature's defense against the continuous attack on the gestation, of gravity and intra-uterine pressure. We must remember that the gestation is, in a sense, a foreign body. It has arrived without warning, the uterus being given only some 5 days to prepare for its reception. It grows with remarkable rapidity, and yet almost no attempt is made by nature to bolster up the musculature of the lower uterine segment, by hyperplasia or by hypertrophy.

The intra-uterine mechanism of the gestation, membranes, amniotic fluid, fetal suspension, etc., is too well known for elaboration here. It serves a great purpose in its demand for an even distribution of pressure over a large area, but it is not enough in itself to offset hernia of uterine content. The mighty factor is the smooth muscle tone phenomenon manifested in the region of the internal os and termed Hegar's sign.

The old contention that the elastic tissue in the cervix had much to do with its sphincter properties was made the object of study. Elastic tissue is found in some abundance around the arteries, to a less extent around the veins and very occasionally between the muscle fibers. It was found in just about the same quantity in the corpus as in the cervix. The cervix of the pregnant uterus showed apparently no more than the cervix of the nonpregnant uterus. It was felt that the elastic tissue content of the cervix was of importance only from a circulatory standpoint.

A THEORY

That the remarkable extent and duration of smooth muscle tone is almost unlimited is shown by the action of the adductor muscle of the bivalve mollusc. Such strength is

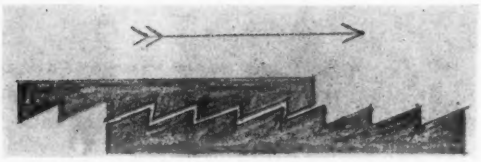


Fig. 1. Diagram to illustrate a catch and ratchet mechanism. The upper piece can be pushed in the direction of the arrow and the total length of the model shortened in this way, but the upper piece cannot be moved back again unless the two pieces are intentionally separated. (From Bayliss.)

known to every one who has opened an oyster. The exact figures are irrelevant; sufficient be it to say that this muscle, one-third square centimeter in cross-section, can withstand a force of pounds over a period of days *without evidence of fatigue* (6).

Consideration of such facts led Grutzner (3) to suggest that the muscle fibers cannot be exerting tensile stress by a continuous excitatory process, but that the fibers must be "hooked up" in some way by a kind of arrangement similar to a ratchet and kept in a position to which the shortening process brought them (1). This theory of the catch or ratchet mechanism of smooth muscle is best explained by the accompanying illustration (Fig. 1). It is only a theory, but an interesting one. It explains fully the ability of the cervix to withstand the forces to which it is subjected; yes, and probably ten times their combined forces. That elusive inhibition by which the "catch" is removed is wrapped up in the veil of phenomenon, labor.

It is probable that most surgeons in dilating cervices have tried to hurry the procedure with powerful Goodell or Bossi dilators or weapons of a similar ilk. It is true that these instruments do add speed to the procedure in that one can often dilate a cervix enough to admit a finger or forceps in 1 or 2 minutes. But how many have also experienced in their hurry that sudden sickening relaxation of the cervix, that feeling as though something had torn through or given way. We have had it happen to us and we have seen it happen to one or two of the best known men in the land.

It is here contended that such pressure has been put on the musculature of the cervix as is put on the bivalve mollusc with the oyster knife, that the intricate ratchet mechanism of the muscle has received such pressure as to overcome all opposition and literally tear the mechanism of the cervix to pieces.

Just how much permanent damage has been done is not known but it is probable that the ability of the patient to carry subsequent pregnancies to a successful conclusion is very much diminished, even, it may be, impossible.

With the graduated dilators the cervix is dilated by degrees over a period of time and the musculature responds by degrees as any muscle will. It is a longer procedure, it is true, but surely time is of little account. Nature would take about 1 hour to dilate a cervix to the size of a 25 cent piece. Surely man has the patience to take at least a third of that time.

Regarding amputation of the cervix, if there be anything in our theory, it is very evident that the removal of any quantity of cervical musculature is going to ruin irretrievably the ratchet mechanism and make subsequent pregnancies certain failures.

Two or three facts must, then, be kept in mind regarding (a) the anatomy and histology of cervical glands, (b) the usual pathology of cervical glands, and (c) the most conservative operation that will accomplish two things: (1) the eradication of infective glands and (2) the conservation of the cervical musculature.

a. The cervical endometrium with its numerous branching, racemose glands, has been well exploited in the past. Much emphasis has been placed on the fact of the branching as compared with the simple tubular glands of the corporeal endometrium (as a matter of fact many uterine glands branch). Perhaps much more important are the facts that (1) the glands are racemose, i.e., have blunt, bulbous terminations, resembling grapes; (2) that the glands, taken singly, narrow in lumen as they approach the cervical canal, so resembling roughly a beaker, such as chemists use; (3) that the glands secrete a thick, tenacious mucus which is excreted normally with some difficulty, and that the glands are readily plugged by inflammation, infiltration, and swelling at their gland mouths. In contrast the uterine glands secrete a thin, watery fluid, readily excreted.

b. An important factor to keep in mind is that endocervicitis is confined in great part to the cervical endometrium and the parts immediately adjacent. The racemose termination of the gland is often infected, it is true, but by no means always, and in any case the glands rarely extend beyond a depth of 3 millimeters at the external os, and at the internal os there are very few glands, and the penetration is very shallow. The resulting infection is almost nil, then, in the region of the internal os.

c. It is felt that in view of these factors, the coning out process as first advocated by Sturmdorf and later modified by many, is the operation of choice. It both eradicates and conserves.

The cervix is really not a sphincter. Probably its entire musculature finds origin in the corpus uteri. Systole and diastole of the cervix is, then, identical with that of the corpus, only slightly later in time. It is contended, though, that the cervix has the *properties* of a sphincter, and that is the important thing, after all.

The object of this paper is not to attempt an explanation which will finally decide the wondrous mechanism of the cervix uteri, nor to evolve a panacea for all its ills. It is hoped merely that a little more consideration will be accorded an organ the treatment of which as observed in hospitals in over half a continent, borders on abuse. The cervix is in a position remarkably strategic. It is the gateway to the citadel, the barrier between the aseptic and the ever septic, the dividing line between things mundane and the great unknown. It accepts or rejects the morbid bacteria or the vital sperm seemingly at will. The ovum is not fertilized, or, if it is, the uterus is unable to carry the conception on to maturity. Should we not emphasize again the remarkable position it holds, and the part it plays in the female genital tract. It is only through such knowledge that we acquire the proper reverence and through it the proper attitude.

REFERENCES

1. Bayliss, W. M. Principles of General Physiology, 1920, p. 534.
2. Child, C. G. Sterility and Conception, p. 903.
3. Grutzner, P. Ergeb. Physiol., iii, 12–88.
4. Herman, J. Obst. & Gynæc., Brit. Emp., 1902, Sept.
5. M. J. Austral., 1923, i, 560.
6. Phanas, Jakob. Pfluger's Arch., cxxxiv, 441, 495.
7. Rottenburg and Schwartz. Surg., Gynec. & Obst., xxxix, 639.
8. Sturmdorf, A. Surg., Gynec. & Obst., 1921, xxii, 93.
9. Young, J. Van D. New York M. J., 1921, cxiv, 395.

SARCOMA OF THE UTERUS

Report of a Case with Review of the Literature

By J. C. BUNTEN, M.D., F.A.C.S., Augusta, Kansas

SARCOMA of the uterus is a subject which requires consideration owing to the fact that in the last few years the condition is being found with increasing frequency. This increase is probably due almost entirely to the better diagnostic methods and the more universal pathological study of both gross and microscopical, myomatous growths of the uterus after operation. While I may repeat some of the statements of other writers, the subject is of sufficient importance to warrant the repetition.

In regard to terminology the following names have been used to designate this type of tumor: sarcoma, myosarcoma, leiomyosarcoma, myoma malignum, malignant leiomyoblastoma, malignant leiomyoma, and malignant myoma. I am of the opinion that sarcoma or myosarcoma would be the more appropriate name.

Uterine sarcoma was first described by C. Mayer in 1860 and later confirmed by Virchow in 1867. In 1867 G. Veit devoted a portion of a chapter in his work upon diseases of women to the affection, describing 3 cases of his own, including 1 case of sarcoma of the cervix which is the first on record. In the next 5 years a number of cases were reported

in Germany. In 1894 Williams wrote an excellent article on the subject, collecting all the reported cases up to that time, numbering 144. In April, 1923, Mason again reviewed the literature in a very interesting article. Between 1894 and 1923 about 200 cases were reported. From the date of Mason's article to the present time I find 9 cases reported, including 1 of my own. This makes a total of 353 cases in the literature.

The etiology of sarcoma is at present unknown so there is little to say on this topic. Statistics show the average age of patients with very malignant tumors to be 50 years, and all other types of cancer 40½ years. In the United States in 1916, 23.4 per cent of all deaths in women was due to malignant tumors of the uterus. These were mostly cancer.

Zacherl states that the proportion of non-epithelial malignant tumors of the uterus to carcinoma is 1 to 40. Evans from the Mayo Clinic records for the period from 1910 to 1918, 22 borderline and malignant, non-epithelial tumors of the uterus while there were 873 carcinomata for the same period, which is the same proportion as that stated by Zacherl.

Sarcomata of the uterus may be divided into three groups: (1) those originating in the mucosa; (2) those originating in the parenchyma; (3) those originating from the cervix.

From Knott's study of 118 cases, 33 were of the first class, 46 of the second class, and 29 of the third class. A question of importance is, do sarcomata originate from pre-existing fibroids? Most observers seem to believe they do. Maroney states that diagnosis must be a matter of individual interpretation in suspicious cases. A composite picture of characteristics as presented by several authorities includes the following points:

1. Increase in size of tumor cells, as compared with normal muscle or benign muscle tumor cells.

2. Shorter and plumper cells with nuclei more nearly oval than normal muscle or benign muscle tumor cells; rounded and "vesicular" nuclei.

3. Inequality in size and irregularity in shape and arrangement of the cells.

4. Lack of differentiation of the cells.

5. Unequal staining of nuclei, and deeply staining nuclei.

6. Presence of immense cells (protoplasmic plaques) with hyperchromatic, single, or multiple nuclei (giant cells).

7. Presence of mitotic figures, typical and atypical.

8. Decrease or absence of stroma fibers between the cells.

9. Thinness or absence of vessel walls.

Kelly and Cullen seem to place definite dependence on inequality in the size and increase in the size of the tumor cells, and do not look upon the presence of mitosis as essential to the diagnosis of malignancy in these tumors.

Ewing says the round cell and the giant cell are the most malignant. Proper and Simpson agree, and state that in doubtful cases the absence of mitotic figures is a criterion of a benign tumor. Mallory believes the presence of mitotic figures is definite indication of malignancy.

Evans stated that of the 72 cases reviewed at the Mayo Clinic, the number of mitotic figures present was in direct proportion to the malignancy of the growth. In 13 cases mitosis was a very common finding, averaging 2,200 to 12,000 mitotic figures for each cubic millimeter. Eleven of these patients were known to have had recurrence within 18 months. In 11 cases mitotic figures ran from 200 to 800 for each cubic millimeter, and in the remaining 48 cases few or none were found. In both of the latter groups the end-results were excellent and as far as Evans was able to determine, there had been no recurrences. Masson states that there is no doubt that mitotic figures are a common finding in the more malignant types of sarcoma.

I am sure that when we consider the proportion of myomata that are malignant we will find that a more careful study of these tumors after removal is essential. From the following statistics one can safely state that in about 2 per cent of all myomata malignant changes take place. Sarcomata of the endometrium constitute about one-third of uterine sarcomata. Sarcoma of the wall occurs either in the myometrium (mural) or more often in a

	No. Cases of Myomata	Per cent Malignant
Murphy	1,000	1.3
Boston City Hospital	827	2.9
Mayo Clinic	4,000	1.8
Frank Warner	100	2.0
Miller	9,750	1.96
Lewis	1,518	1.4
Olshausen	6,470	1.2

fibroid. Noble claims that 2 per cent of all fibroids show malignant changes, which agrees with the above table. Melanotic sarcoma is never primary in the uterus.

It is interesting to note the occurrence of combined tumors, that is, a sarcoma and a carcinoma in the uterus of the same case. Moise states that the combined tumors are rare. Virchow speaks of a carcinomatous degeneration of a uterine sarcoma. Palmer Findley, in an article in 1905, discussed the combined malignant tumors of the female genitalia. A more recent article is that of R. H. Jaffe in October, 1923, in which he states that the combination of sarcomatous and carcinomatous tissue may be due to the growing of a carcinoma and a sarcoma into one another, both of them taking origin in different parts of the same organ; or they may arise from the same place. The name sarcocarcinoma should be used only for this latter type of tumor.

There are three possibilities to explain the histogenesis of these tumors: (1) the carcinoma may have been the primary tumor, the stroma of which changed into a sarcoma; (2) the sarcoma may have been followed by the carcinoma; or (3) both tumors may have developed at the same time. Most of the tumors formerly reported as sarcocarcinomata are surely pure carcinoma with partly sarcoma-like appearance of the tumor cells.

I have found 13 such cases reported in the literature, and will mention them as follows:

Case reported by Jaffe.

A round cell sarcoma in the mucous membrane of the corpus. Underneath this tumor were carcinomatous cells. (Gebhard.)

A woman expelled two necrotic tumors the size of a man's fist which were round cell sarcomata. At autopsy the cavity of the uterus contained typical alveolar carcinoma. (Rabl-Ruckhard.)

Currettage revealed an alveolar carcinoma and 2 days later there was expelled a tumor the size of a goose-egg, which was a vascular spindle cell sarcoma. There was also a fibroid and a polyp of the corpus and cervix. (Rabl-Ruckhard.)

A round cell sarcoma of the corpus the size of a fist was removed and recurred. There were carcinoma cells in the tumor. (Klein.)

A sarcomatous polyp of the corpus. Over the remaining surface of the corpus was an alveolar carcinoma. (Emanuel.)

A sarcomatous polyp in the corpus (round cell). In the upper segment of the cervix was an adenocarcinoma. (Von Franque.)

A spindle cell sarcoma the size of a walnut in the corpus. Higher in the fundus was an adenocarcinoma. (E. Opitz.)

A sarcomatous polyp of the cervix and adenocarcinoma of the corpus. (Amann.)

Degenerated sarcoma with multiple carcinoma growths. (Lagraze.)

An adenofibroma of the uterus, the upper portion of which was carcinoma and the lower portion sarcoma. (Ivanoff.)

Carcinomatous degeneration of a cervical polyp. Hysterectomy was followed by a sarcomatous growth at the base of the broad ligament. (P. Findley.)

I wish also to make mention of the article by Taussig in 1914, in which he had reviewed the literature up to that time and found that out of 141 cases of tumors of the round ligaments, 5 were sarcoma.

SIGNS AND SYMPTOMS

From the standpoint of diagnosis there is nothing in the history or physical examination which makes it possible to suspect malignancy except in advanced cases where operative cure is hopeless. Distant metastases do not seem to occur in these cases. Recurrences are all local. The symptoms are not definite. In general they are those of carcinoma. A watery or blood-tinged vaginal discharge, bleeding, menorrhagia, and metrorrhagia occur in one-half of the cases. Other evidences are menorrhagia during the menopause; pain in the tumor independent of menstruation; and an abundant, thin, watery discharge after the menopause which does not lessen with the progress of the disease. Soft masses may escape in large quantities from the uterus. In adolescents the youth of the patient may be suggestive as myoma in these cases would be rare. The uterus is softer and less resistant; it increases in size more rapidly and to greater degree than with carcinoma. Pain is more prominent than in carcinoma.

There is possibly more profuse bleeding and less breaking down than with carcinoma. Metastases are less common, and occur later than with carcinoma. Sarcoma is more common after than during menstrual life.

TREATMENT

The only logical treatment in these cases is the early and complete removal of the uterus. It is claimed that roentgenotherapy alone has given some good results, and there are cases which are not good surgical risks in which it would be the treatment of choice. However in the large majority of cases surgery is preferable, especially since the mortality in these operations is very low.

REPORT OF CASE

Mrs. J. A. T., age 27, white, married, on April 17, 1921, began to menstruate and continued until the 26th. She has been feeling a little weak for the last 3 weeks. The flow started again May 4 and lasted to the 9th. On May 26 the flow began and continued until June 6. September 23 the flow began again and continued until October 13. The amount of the flow at these times was always quite profuse, with moderate leucorrhœa between times. Her last baby was born in January, 1921, the flow lasting for six weeks following delivery. She has had 7 children, one pair of twins. Menses have always been fairly regular between pregnancies. She has some swelling of the ankles, and occipital headaches. The stomach is normal, appetite good. She sleeps fairly well and the bowels are regular. The kidneys are normal. There is no cough. The past history is negative except for the usual diseases of childhood.

Physical examination revealed a patient somewhat anæmic and weak. The tonsils were not good and the teeth were only fair. Lungs and heart were normal. The pulse was a little rapid but of good quality. The abdomen was negative. Vaginal examination revealed an old second degree perineal laceration and bilateral cervical laceration. The uterus was retroverted and the supports very much relaxed. There was a bloody discharge from the cervix, no particular tenderness about the tubes or ovaries. The fundus of the uterus seemed abnormally hard and a little irregular. There was slight œdema of the legs. The reflexes were normal. The urine was straw colored, acid, 1038, no albumen, no sugar, microscopically negative.

A diagnosis of fibroid uterus was made.

Hysterectomy was performed October 13, 1921. The patient made an uneventful recovery and has remained well up to the present time.

Pathological report. In the interior of the fundus near one cornu there was a dark colored tumor the size of a walnut. The base of this tumor was composed of what appeared to be a new growth of muscular and fibrous tissue, but the growth does not have the ear-marks of fibromyoma tissue, and appears to be indefinite in character.

The superficial part of the mass shows much evidence of chronic inflammation and considerable erosion. This is greatest in the neighborhood of the hæmorrhagic area, and at this place there are definite cells, and blood sinuses without walls. This is a malignant sarcoma, though undoubtedly in an early stage of malignancy.

BIBLIOGRAPHY

AMANN. Ueber Neubildungen der Cervicalportio des Uterus. Munich, 1892.

EMANUEL. Ueber gleichzeitiges Vorkommen von Carcinom und Sarkom im Uteruskorper. Ztschr. f. Geburtsh. u. Gynaek., xxxiv, 1.

EVANS, N. Malignant myomata and related tumors of the uterus. Surg., Gynec. & Obst., 1920, xxx, 225–239.

FARR, C. E. Sarcoma of the uterus. Ann. Surg., 1924, November.

FINDLEY, P. Combined malignant tumors of the female. Surg., Gynec. & Obst., 1905, i, 350–353.

FULLERTON, W. D. Uterine sarcoma. Surg., Gynec. & Obst., 1914, xix, 711–718.

GEBHARD. Path. Anat. d. weibl. Sexualorgane. Leipzig, 1899.

GARDNER, L. U. A case of metastatic leiomyosarcoma, primary in the uterus. J. Med. Research, Boston, 1917, xxxi, n.s., 19–29.

IWANHOFF. Drusiges, cystenhaltiges Uterusfibromyom, complicirt durch Sarkom und Carcinom. Monatschr. f. Geburtsh. u. Gynaek., vii, 295.

JAFFE, R. H. Sarco-carcinoma of the uterus. Surg., Gynec. & Obst., 1923, xxxvii, 472–475.

KLEIN, G. Uterussarcom. Muenchen med. Wchnschr., 1890, 170.

KNOTT, VAN BUREN. Ann. Surg., 1901, xxxiii, 137.

MARONEY, W. J. Sarcomatous change in uterine fibroids. Am. J. Obst., 1916, lxxvii, 445–450.

MASIN, J. C. Sarcoma of the uterus. Am. J. Obst. & Gynec., 1923, v, 345.

MILLER, J. R. The relation between sarcoma and myoma of the uterus and its bearing on X-ray therapy of uterine myomata. Surg., Gynec. & Obst., 1913, xvi, 315–321.

MILLS, H. W. Sarcomatous degeneration of uterine fibroids. Report of two cases. Interstate M. J., xxv, No. 3.

MOISE, T. S. An adenocarcinoma and a round cell sarcoma arising in the same uterus. Surg., Gynec. & Obst., 1924, 83–87, July.

OPITZ, E. Ztschr. f. Gebursh. u. Gynaek., 1899, xlix, Heft 2.

PERLSTEIN, I. Mesodermal mixed tumors of the uterus; report of a case of botryoid chondrosarcoma of the endometrium. Surg., Gynec. & Obst., 1919, xxviii, 43–55.

RABL-RUCKHARD. Beitr. z. Geburtsh. u. Gynaek., 1872, i; also Arch. f. path. Anat., xcii, 191.

TAUSIG, F. J. Sarcoma of the round ligament of the uterus. Surg., Gynec. & Obst., 1914, xix, 218–223.

VIRCHOW. Die krankhaften Geschwuelste, ii, 351.

VON FRANQUE. Carcino-sarkom Endothelioma Tubae. Ztschr., xlvii, 211.

Idem. Ztschr., xii, Heft. 2

WARNER, F. Uterine fibroids with reference to their tendency to become sarcomatous. Am. J. Obst., 1917, lxxvi, No. 6.

OSTEITIS FIBROSA CYSTICA

Report of Six Cases

By J. NEWTON SISK, M.D., Madison, Wisconsin
Section on Roentgenology, Jackson Clinic

THE writers on osteitis fibrosa cystica are in accord that this condition is a distinct, benign, central lesion of the bone. The uniformly good results obtained by conservative surgery in cases of osteitis fibrosa cystica make this an important disease to be considered in cases of central bone lesion of questionable clinical course.

HISTORY

Our knowledge of osteitis fibrosa cystica dates back to 1891, when von Recklinghausen described in detail a fibrocystic degeneration in the long bones, to which disease his name has since been attached. Virchow, in 1876, described a cyst of the humerus, reporting liquefaction of a chondroma as the cause. Later Rindfleisch (1886) and Hirschberg (1889) described cysts occurring in osteomalacia. This early material was reported from necropsy findings, the X-ray not being used in the study of these conditions, according to Meyerding, until 1903, when Heineke reported its use in his cases.

The literature on fibrocystic disease is comparatively recent, but has been accumulating rapidly since 1900, the increasing interest being stimulated largely by the findings in X-ray examinations. Contributions to the recent literature have been made by Beck, Murphy, Muller, Silver, Freiberg, Ashhurst, Brown, DeCourcy, Bloodgood, Barrie, Percy, Platou, Lotsch, Landon, Boyd, Bullowa, Meyerding, and several others. In volume, the work of Bloodgood exceeds that of the others.

Although the distribution of cases does not observe any geographical limits, the reports of Brown, Elmslie, Landon, Davies, Boyd, and other European contemporaries lead to the conclusion that the occurrence of osteitis fibrosa cystica has been less frequently observed on the other side of the Atlantic than in America.

Osteitis fibrosa cystica is most commonly recognized as a local disease, but it may be general. Ashhurst, Bloodgood, and Meyerding report cases in which the lesions were located in more than one bone. The most common sites for the local lesions are the femur, humerus, and tibia, in the ratio, according to Meyerding, of 6:5:3. Thus far, no case has been reported in which the ribs or skull were involved. Potts and Hatton, and Galvin each reported one case in which the lesion was located in the mandible. Bloodgood's case, in which the focus was in the spinous process of a vertebra, is the only one reported with involvement of any part of the vertebral column (6).

ETIOLOGY

The earliest idea (von Recklinghausen) was that the process is basically an inflammatory condition; that is, a chronic productive inflammation, whereby fibrous tissue is substituted for bone structure. While it may rest on an inflammatory basis in its early etiology, the lesion at operation shows none of the accepted signs of inflammation. According to Ewing, Rehn, and several other able pathologists continue to base their conclusions on this hypothesis of inflammatory origin. In an effort to work out the etiology some men have been led, by the evidence of the malacia, to attempt to show a relation between von Recklinghausen's disease and Paget's disease in the adult, and rickets in children. While these conditions unquestionably have in common some fundamental change in calcium metabolism, some similarity in deformity and radiographic appearance, plausible evidence linking the causes of these malacias is yet to be established. All the theories of endocrine dyscrasia in relation to bone metabolism, and the presence of a filterable virus, have been considered with disappointing results. Trauma is at present properly considered a most conspicuous factor in the etiology.

PATHOLOGY

The pathogenesis throughout the course of the disease is the picture of metaplasia characterized by early malacia (osteodystrophia juvenilis?). At operation no changes in the soft parts are observed grossly when the incision is made to expose the bone lesion. The periosteum is quite easily freed from the bone. A gray, almost white cortex is revealed, with the appearance of a structure the vitality of which is lowered. When the bone surface is scratched with a curette, there is not that sensation of "dragging back" on the curette noted in normal bone. The cortex is easily crushed. When the bone canopy is penetrated the contents are usually found to be fluid, thin or gelatinous, and yellow or reddish in color, according to the amount of blood present.

Microscopic evidence of metaplasia has cast suspicion on the benignity of the disease. Witness the remarks of Bloodgood (5) that in almost all of his cases of bone cysts with osteitis fibrosa he has found cellular areas with spindle cells or round cells. The spindle cells are apparently of the connective-tissue type which ultimately form fibroblasts and fibrous tissue, and the round cells are either osteoblasts which have not yet formed bone, or cells which arise from proliferation of the endothelial cells of blood vessels. The microscope shows a tissue made up of cellular fibroblastic framework with delicate bone trabeculæ embedded in it, and if the lesion is near the epiphysis, a circumscribed area of hyaline cartilage may be found, even grossly. The finding of hyaline cartilage in these cysts probably gave Virchow his basis for concluding that osteitis fibrosa cystica resulted from liquefaction of a chondroma. Bloodgood (4) hints that the finding of the cystic area near the epiphysis suggests the likelihood of discovering cartilage in the lesion; also the cartilage is not present in sufficient quantity to justify the conclusion of an original cartilage mass. The walls of old lesions are lined with a fairly dense connective-tissue membrane, the fibers of which are concentrically arranged and bounded by short strands. No true endothelial or epithelial lining is present. The fibrils of connective tissue have few nuclei, and only a moderate number of blood vessels are seen throughout the fibrous portion of the mass. Some areas are infiltrated with round cells and resemble granulation tissue which may be confused microscopically with small round cell sarcoma. Resorption of marrow and bone goes on with this new formation, differing from the normal in that osteoclastic giant cells often remain and may gather in clumps. Perhaps because of some defect in calcium deposit, the usual course is that the tissue remains in the osteoid stage instead of going on to form normal bone. True malacia occurs. In patients who recover spontaneously, or who recover after non-surgical treatment, as has been reported (7), or those in whom the bone structure appears normal in the X-ray examination after a long period, the probable defect with respect to calcium metabolism has been corrected, and the bone-forming process has resumed its normal function.

DIAGNOSIS

The result of X-ray examination, while unsupported, is not conclusive, yet it is reasonably characteristic, and is the most reliable aid to diagnosis. Murphy (25), as early as 1913, remarked that in cases of fibrocystic disease, the diagnosis may as well be made by X-ray examination as with the microscope. As a matter of common experience, the surgeon who depends upon frozen sections for diagnosis in bone tumors of central origin will come to grief. For example, in a number of Bloodgood's reported cases in which tissue was sent to the laboratory for study, a diagnosis of sarcoma was made from the frozen section, while further study and the patient's clinical course showed the lesion to be benign. The basis of the primary report was presumably the misinterpretation of metaplastic forms for those of malignancy.

Acute bone lesions with definite etiology give little difficulty in diagnosis and are not considered in this discussion. The central bone tumors with cyst formation to be considered in making a presumptive diagnosis are osteitis fibrosa cystica, giant-cell sarcoma, myxoma, chondroma, and sarcoma. These are eliminated by the rules of probability, the clinical facts, and X-ray findings. The

pre-operative findings are of no value in deciding the consistency of the lesion, whether liquid, solid, or lined with a membrane, but that is not essentially important. Reliable pre-operative evidence enables the surgeon to make a presumptive estimate of how extensive his work is going to be, and to make a reasonable prognosis concerning the benefit the patient will gain from an operation.

Myxoma and chondroma of bone are equally rare, comprising together about 8 per cent of all central bone tumors. Although the site of predilection is the bones of the hands or feet, other bones may be affected. Ewing writes with regard to chondroma: "The X-ray shows osteoporosis of the ends of the bones, and often a cystic appearance, while the compact bone of the ends of the shaft may be very deficient. At various points, usually about the joints, the multiple outgrowths appear. The structure shows a persistence and overgrowth of poorly ossified or calcified cartilage, in which the cells are irregular in size and form (Carmen, Fisher). The ordinary epiphyseal line is irregular or obliterated." The central myxoma absorbs the shaft and periosteum and invades the soft tissues. Osteitis fibrosa cystica does not manifest itself by any change in periosteum as a rule, and never reveals more than slight thickening, the structural change being confined within the cortex of the shaft. The lesion is more often metaphyseal, and is never epiphyseal.

Between osteitis fibrosa cystica and giant cell sarcoma the difference can as easily be shown by the X-ray as with the microscope. Some points of diagnostic importance in this particular are: the longitudinal extent of enlargement of the shaft is more limited in giant cell sarcoma, and the distention is greater. In giant cell sarcoma there is usually no bowing of the shaft; in osteitis fibrosa cystica bowing is common. The cystic pockets in osteitis fibrosa cystica are separated by trabeculæ of compact bone and are clean cut, differing in this respect from sarcoma. The surrounding tissue is not involved in osteitis fibrosa cystica.

The onset and length of the history and the frequency of occurrence are a valuable help in differentiating osteitis fibrosa cystica and giant cell sarcoma. Osteitis fibrosa cystica is the most common non-malignant lesion of bone (26). Its onset is usually in childhood, while in giant cell sarcoma the onset is usually after 20 years of age. A few cases have been reported with an earlier history, yet there are still fewer cases of osteitis fibrosa cystica with a history of onset after 20 years. Pain may not be severe in giant cell sarcoma but is usually present, and swelling generally occurs sometime after the appearance of pain; in osteitis fibrosa cystica, pain is not a conspicuous feature, unless there has been a fracture. Pathological fracture is common in osteitis fibrosa cystica, less so in giant cell sarcoma. The more malignant central sarcoma is at once recognized by its late history, its destruction of the cortex, involvement of the periosteum and surrounding tissue, and the parchment crackle elicited on palpation of the tumor. Expansion of the shaft as shown by X-ray examination of the benign giant cell sarcoma is missing in the malignant type (16).

Next to the roentgenologic picture in point of diagnostic importance stands the clinical picture of osteitis fibrosa cystica, which, in most cases, is uniform and of long duration. The onset is in early life. There is usually history of trauma, swelling, and perhaps deformity. Pain, if present, is rarely severe at any time, and disability is not noted, unless there is a fracture. Pathological fracture is common, frequently recurrent, and is the turn of affairs that usually leads to discovery of the disease.

TREATMENT

In the treatment of osteitis fibrosa cystica, the greatest economy of time is served by conservative surgery at the time the lesion is discovered. If the patient is confined to bed many months, some of these lesions heal spontaneously, as was suggested by Bloodgood (7) recently. The uniform success obtained by surgery, with a comparatively short convalescent period, however, argues against the justice of so liberal a disposition of the patient's time by the attending surgeon, even if the indications for this watchful waiting treatment are present. If the cystic area is small, it is sufficient to expose the tumor, break through the thin canopy, and thoroughly clean out all soft tissue with a curette. Follow-

ing this procedure the tumor stops its growth, just as do benign cysts of other tissue when their contents are evacuated and the cystic capsule is removed. The cortical canopy should be crushed in, and if bone chips are introduced into the cavity, healing is probably hastened, as in Case 6 herein reported. These chips are easily obtained with a trephine from the surrounding normal bone. Beck's paste is contra-indicated in these cases, as it forms an unabsorbable irritant. The Moorhof bone plug is also of little use in such cases; moreover, the difficulties attending its application argue against its employment. If proper precautions are taken to prevent infection, the average lesion will heal by primary union. The time for new bone to fill the cavity compactly is not great, one to three and one-half months, depending on the extent of the lesion. If the lesion is unusually extensive and there is a desire to correct or prevent deformity, much aid may be necessary to insure a useful member. To accomplish this it is frequently necessary to introduce an implant from the tibia, as was done in our Cases 1, 2, and 3. It is important to take this implant from the tibia of the unaffected leg when the lesion is in the lower extremity, as a Buck's extension, necessary to prevent the displacement or destruction of the implant by muscular contraction, must not be applied over the skin wounds. Except in cases of extensive involvement or deformity, efforts at plastic surgery are not indicated. In Case 4 no other operative procedure was employed than removal of the tumor *in toto;* a cast was applied as a precaution, since the patient was going from under our care. A criticism against curetting these lesions is made by DeCourcy on account of the difficulty in ruling out sarcoma. Sarcoma with a history of as long duration as that presented in cases of osteitis fibrosa cystica would have a rather typical picture.

REPORT OF CASES

Cases 1, 2, and 3 are presented because of their characteristic clinical histories of bone cyst and because of the excellent surgical results obtained.

CASE 1, No. 4594. F. P., a schoolboy, age 14 years, came to the Jackson Clinic September 21, 1916, on account of pain and disability in the right hip following a fall the previous day. There was some swelling in the right thigh, pain on manipulation, and crepitation could be felt. There was an elevation of temperature.

X-ray examination revealed a pathological fracture immediately below the trochanters, also a central fibrocystic degeneration of the upper third, characteristic of osteitis fibrosa cystica (Fig. 1). Other laboratory examinations were negative.

It is important to note that in 1914 the patient noticed slight pain in his hip on walking. At that time, also, there was some elevation of temperature; but there was no history of trauma, and the general health was good.

The patient was operated on September 22, 1916. A lateral incision beginning immediately below the greater trochanter and extending downward 24 centimeters exposed a normal periosteum. This was incised and easily stripped from the cortex, which was grayish white and quite brittle. Scratching with a curette did not give the sensation of normal bone. The lateral aspect of the canopy was removed above and below the fracture, which was immediately below the proximal limits of the fibrocystic change. The contents of the numerous cystic areas was scanty (there was some evidence of hæmorrhage at the site of fracture) and of a thick fluid consistency, rather reddish yellow in color. There was abundant evidence of a fibrous membrane in the vacuolated upper third, which led the surgeon to confirm the opinion of the roentgenologist that this was a case of osteitis fibrosa cystica. After the cavity had been thoroughly cleaned with the curette, an inlay graft 18 centimeters long was placed in a lateral groove uniting the upper and lower fragments. A cast, with extension to prevent destruction of the inlay by muscular contraction, was applied and kept on for 4 weeks. Dressings were applied through a window in the cast for 2 weeks until the wound had healed by first intention. The cast was changed twice, being discontinued at the end of the ninth week when union appeared to be good. The recovery was complete.

An X-ray examination in 1924, 8 years after operation, revealed a femur, the bone structure of which varies not at all from the normal (Fig. 2). There exists only a slight coxa valga to suggest that the femur has ever been other than normal.

CASE 2, No. 4600. R. M., a schoolboy, age 15, came to the Clinic September 23, 1916, complaining of disability of the right arm. On the previous day while playing baseball he was in the act of making a long throw when he heard something snap, after which he was unable to raise his right arm, or put it behind his back. There was some swelling and pain, and on manipulation, crepitation could be felt in the upper third of the right humerus.

Clinically, a diagnosis of pathological fracture was made. X-ray examination warranted a conclusive diagnosis of osteitis fibrosa cystica (Fig. 3).

The boy was admitted to the hospital the following day. The lateral aspect of the arm was laid open,

Fig. 1 (left). Case 1. Osteitis fibrosa cystica of upper third of femur; before operation.

Fig. 2. Case 1. Roentgenogram taken 8 years after operation.

Fig. 3 (left). Case 2. Osteitis fibrosa cystica of upper third of humerus; before operation.
Fig. 4. Case 2. Patient 7 years after operation.

Fig. 5 (left). Case 3. Osteitis fibrosa cystica of upper third of humerus; before operation.
Fig. 6. Case 3. Patient 4 years after surgical repair.

Fig. 7 (left). Case 4. Localized osteitis fibrosa cystica of medial aspect of proximal end of tibia.
Fig. 8. Case 4. Lateral view of patient in Figure 7.

the incision revealing a gray, vitiated cortex which crushed easily. The contents of the cystic area was gelatinous and, at the site of the fracture, considerably blood-streaked due to hæmorrhage. The contents were removed with a curette. A definite fibrous membrane lined the numerous cystic spaces. A long bone graft from the fibula was laid into the groove, uniting the fragments of the humerus. A cast, in which a window was cut for dressing, was applied to the arm and chest. Extension was applied by weights and pulleys to counteract muscular contraction. At the end of 2 weeks the incision was healed. Six weeks later callus was laid down along the entire transplant, but a new cast was applied for an additional 4 weeks. At the end of 6 months from the time of operation good function was restored in the member.

In 1923, 7 years after operation, the X-ray examination showed a humerus with slight deformity as to shape, otherwise normal (Fig. 4).

CASE 3, No. 33296. Miss E. K., age 12, was brought to the Jackson Clinic October 2, 1919. The chief complaint was partial paralysis of the right arm following an injury 4 days previously. The history dated from 1911 when the patient was 2 years old. She had fallen and injured the right shoulder after which she had been unable to raise her arm over her head or put it behind her back for a few days. During the 2 years preceding her first visit to the Clinic, she had fallen at play four or five times, injuring the right shoulder. After each of these injuries she had suffered a partial paralysis of the arm.

On examination the right shoulder was noted to be swollen, tender, and painful on manipulation. Crepitation could be felt at the upper third of the humerus. A clinical diagnosis was made of bone cyst with recurrent fracture. X-ray examination confirmed the clinical findings and led to a diagnosis of osteitis fibrosa cystica of the upper third of the right humerus (Fig. 5).

The patient entered the hospital October 23, 1919. The following day the arm was opened with a lateral incision at the upper third. The cystic area was curetted, and an Albee "inlay-graft" from the tibia was inserted. The patient apparently recovered and was discharged from the hospital after 5 weeks.

An X-ray examination 4 years after surgical repair showed a practically normal humerus (Fig. 6). Close inspection revealed no deformity and there was no limitation of function.

CASE 4, No. 30635. The principal interest in this case is the occurrence of early osteitis fibrosa cystica with chronic osteomyelitis in a different and remote member.

Miss M. P.., age 16, came to the Jackson Clinic May 7, 1921. She had had pneumonia at 3 years of age and rheumatic fever at 15. Her chief complaint was chronic pain, swelling, and disability of the left foot of 14 years' duration. The foot had been lanced when she was 6, 11, and 14 years old. A large amount of pus had been removed each time, after which procedure the member had apparently healed, but each time had become swollen and painful again in about 6 months.

Besides a low grade inflammatory condition of the left foot, physical examination revealed a slight asymmetry on the lateral aspect of the right leg immediately below the knee. On palpation, this area appeared to be a hard, smooth tumor attached to the tibia. The tumor was slightly tender to deep pressure and had recently given slight pain. There was no superficial evidence of an inflammatory condition at this site, and no history of injury.

The X-ray examination revealed, besides an osteomyelitis confined to the bones of the foot, a rarefied area in the head of the right tibia, resembling a trabeculated cyst (Figs. 7 and 8). Three centimeters below the cyst was a small sequestrum and a small sinus which did not appear to communicate with the cyst. Diagnosis was made of osteitis fibrosa cystica in the head of the right tibia complicating an old osteomyelitis of the left foot.

Laboratory examinations at this time showed a trace of albumin in the urine, hæmoglobin 75 per cent, leucocytes 17,650, and a negative Wassermann reaction on the blood.

The patient entered the hospital May 16. Necrotic foci of osteomyelitis were removed from the left foot. Also surgical attention was given to the cystic area in the tibia. A semicircular flap was turned from over the swelling on the upper end of the right tibia exposing a vitiated cortex. A small sequestrum, which lay at the lower end of the incision, was removed. The bone canopy of the cyst

Fig. 9 (left). Case 5. Lateral view, osteitis fibrosa cystica of the bones of the face and skull. An enormous cystic projection is noted on the front of the face.

Fig. 10. Case 5. Postero-anterior view, case 5. Large fibrocystic area of density of right maxillary region is shown.

was punctured, the soft material removed, and the cavity thoroughly curetted. The margins of the cavity were then flattened to meet the soft parts and closure was made with plain gut without drainage. Concerning the findings at this site the notes on the surgical record read: "The cyst is trabeculated and lined with a membrane. It does not resemble osteomyelitis. There is no apparent tendency toward bone proliferation and no definite necrosis, but there is rather the appearance of a rarefying osteitis."

The curettings from the cyst were submitted to Dr. Bunting of the Pathological Department of the University of Wisconsin Medical School, and he reported.

"The bone in general shows rarefaction, though an occasional trabecula appears thickened. The marrow between the trabeculæ is in general fatty and contains large thin-walled vessels from which there has been old and recent hæmorrhage (the former shown by blood pigment in the phagocytic cells). There are some larger hæmorrhages in process of organization. The wall proper of the cyst consists of a fibromyxomatous tissue which does not suggest a neoplasm, but rather an atypical inflammatory tissue or a tissue resulting from organization. There are practically no indications of an inflammatory process, as such, in the specimen, and one gains the impression that hæmorrhage might have been the primary lesion. This is difficult to establish because there have been later hæmorrhages into the newly formed tissue. The lesion then may have been primarily inflammatory leading to injury to the vascular endothelium and hæmorrhages, and the organization of these led to covering up the early process."

A plaster cast was fitted to the right leg and thigh, and hot boric acid packs were applied to the left foot. After a week of this treatment the patient was allowed to return home with instructions to have the cast removed in 3 weeks. At the time of discharge the infection in the foot was markedly improved.

At our request this patient returned for observation March 9, 1924. She has had an occasional flare-up of osteomyelitis in the left foot, but is in good health in all other respects. X-ray examination made at this time showed a satisfactory healing of the cyst in the tibia.

Case 5, No. 33215. Osteitis fibrosa cystica does not commonly occur in the flat bones. The principal interest in this case lies in the location of the cystic process, and in the excellent results obtained from surgical treatment.

Mr. J. G., age 25 years, came to the Jackson Clinic November 17, 1922, on account of a marked deformity on the right side of the face. This gave him a peculiarly ludicrous appearance, to his great discomfiture. The history dated from a fall when he was 3 years of age. An enormous growth had appeared soon afterward on his right cheek and had not changed in size since that time. No interference with breathing had been noted.

Slight pressure produced some pain. When the nose and mouth were examined, slight bulging of the lateral wall of the right nasal chamber was observed, with a marked intra-oral bulging of the right side of the hard palate. The right upper molars were widely separated by pressure from the growth. The right antrum was opaque to transillumination.

The report of an X-ray examination (Figs. 9 and 10) made at this time reads:

Fig. 11. Case 5. Lateral view after removal of the facial tumor.

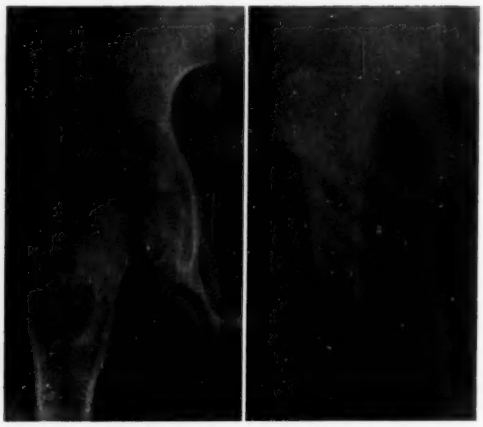


Fig. 12 (left). Case 6. Localized osteitis fibrosa cystica in the proximal end of the right femur; before operation.
Fig. 13. Case 6. One year after curettement and implantation of bone chips.

"This case is characterized by the existence of three areas of irregularly rarefied bone confined to the right side of the face and skull. The area of greatest absorption is in the facial bones and is surrounded by a ring-like periphery of sclerosed bone. All areas of absorption, however, are in a greater or lesser degree subdivided into vacuoles. In addition to the cystic appearance, there is much increase in density of adjacent bony structures. Here and there, especially in the facial bones the fibrous density is accompanied by cystic degeneration and the two processes exist apparently in equal degree. After the exclusion of lues, giant-celled sarcoma, and Paget's, the diagnosis becomes probably one of osteitis fibrosa cystica (von Recklinghausen's disease)."

Examinations of the blood and urine were negative.

The patient entered the hospital November 20, 1922. Since the desire of the patient was for the correction of his facial deformity, no measures were taken against the cystic areas in the bones of the skull. An incision was made immediately below the outer canthus of the right eye extending medially to the border of the nose and then downward, dividing the upper lip. Skin and muscles were reflected *en masse*. A bony cystic area, 5 by 8 centimeters, involving the upper right side of the face, was chiseled away, and the normal contour of the face was established. Closure was made with drainage. The postoperative examination of the tissue removed, confirmed the diagnosis of osteitis fibrosa cystica.

Except for a scar at the site of the incision, there is but slight evidence of the former deformity. The cosmetic result in this case has justified the measure taken.

Case 6, No. 38148. Miss R. S., age 8 years, came to the Jackson Clinic November 14, 1923. Her chief complaint was a limp and pain in the right hip when she walked. The patient's condition had first come to the attention of the mother in the latter part of 1922 when a slight limp had been noted on the right side. This had gradually become worse, and the child had complained of some pain on walking. No history of loss of weight, night cries, cough, elevation of temperature, or trauma could be elicited. The patient's general health has remained good.

On the basis of an X-ray examination made elsewhere, October 14, 1923, a diagnosis of tuberculosis had been made and a plaster cast had been applied at that time. The cast was removed November 3, when another X-ray examination was made by the same doctor. The lesion had again been called tuberculosis of the femur and an operation had been advised.

A physical examination made in this Clinic November 14, disclosed no other information than slight pain on manipulation of the right hip. X-ray examination revealed a polycystic condition between the trochanters of the right femur which had the characteristics of a local lesion of osteitis fibrosa cystica.

The patient was admitted to the hospital the following day. An incision was made over the outer aspect of the greater trochanter and carried downward 15 centimeters. With a trephine the wall of the cyst was penetrated and the contents curetted. The cortical canopy was then crushed in, and the remaining cavity filled with bone chips obtained with a trephine from the femur 5 centimeters below the cavity. The periosteum was closed separately, then the soft parts, without drainage. A cast applied to the right hip and pelvis was left on for 4 weeks. The surgeon reported the cyst to have had a membranous lining, with other characteristics of cystic osteitis fibrosa.

The patient has been kept under observation; the last X-ray examination October 10, 1924, showed a satisfactory course.

SUMMARY

1. Osteitis fibrosa cystica is a distinct, benign, central bone disease, the history of which has been developed since 1891.

2. The etiology is not definitely known; but trauma appears to be a common factor at the time of diagnosis.

3. The pathogenesis appears to be that of metaplasia and early malacia.

4. Diagnosis is made by the X-ray examination as first aid, supported by a long history with onset in early life, and, if there is no fracture, little or no loss of function. Differential diagnosis is to be made between osteitis fibrosa cystica and other common central bone lesions. Microscopic examination of tissue removed is essentially important in making a prognosis if the signs of benignity or malignancy are definite; otherwise, the postoperative clinical course is the only dependable basis on which to form conclusions.

5. The treatment is distinctly surgical at the time the lesion is recognized.

6. A review of the cases herewith presented supports the conclusions above enumerated.

REFERENCES

1. Ashhurst, A. P. C., Bromer, R. S., and White, C. Y. Cystic disease of the bones; a study of fifteen cases. Arch. Surg., 1923, vi, 661–730.
2. Barrie, G. Fibrocystic and cystic lesions in bone. Ann. Surg., 1918, lxvii, 354–363.
3. Beck, C. Osseous cysts of the tibia. Am. J. M. Sc., 1901, cxxi, 666–671.
4. Bloodgood, J. C. Benign bone cysts, osteitis fibrosa, giant cell sarcoma and bone aneurysm of the long pipe bones. Ann. Surg., 1910, lii, 145–189.
5. Idem. The diagnosis and treatment of benign and malignant tumors of bone. J. Radiol., 1920, i, 147–328.
6. Idem. Bone cysts (osteitis fibrosa): variety-polycystic osteitis fibrosa. J. Radiol., 1923, iv, 1–9.
7. Idem. Benign bone cysts (osteitis fibrosa) acetabular cavity upper end of the femur, trochanters, neck of head. J. Radiol., 1924, v, 3–9.
8. Boyd, S. A case of "fibrocystic disease" of the femur. Proc. Roy. Soc. Med., London, 1914–1915, viii, Clin. Sect., 35–40.
9. Brown, K. P. Solitary cyst in the humerus. Edinburgh M. J., 1923, xxix, 145–147.
10. Bullowa, J. G. M. Osteitis fibrosa. Med. Rec., 1915, lxxxvii, 539.
11. Carman, R. D., and Fisher, A. O. Quoted by Ewing, loc. cit.
12. Davies, H. M. Osteitis fibrosa cystica. Proc. Roy. Soc. Med., London, 1914–1915, viii, Clin. Sect., 34.
13. DeCourcy, J. L. Osteitis fibrosa. J. Am. M. Ass., 1919, lxxii, 1612.
14. Elmslie, R. C. Fibrous and fibrocystic osteitis. Brit. M. J., 1912, ii, 1367–1369.
5. Idem. Fibrocystic disease of the bones; benign cysts, osteitis fibrosa with formation of tumors and giant cell sarcomata (von Recklinghausen), and mollities ossium with giant-cell sarcomata. Brit. J. Surg., 1914, ii, 17–67.
16. Ewing, J. Neoplastic Diseases. Philadelphia: W. B. Saunders, 1922, p. 1054.
17. Freiberg, A. H. Some diagnostic features of certain intra-osseous lesions, osteitis fibrosa, bone cyst, and their relation to intra-osseous lesions. Lancet-Clin., 1913, cix, 504–511.
18. Galvin, A. H. Osteitis fibrosa cystica occurring in a flat bone. California St. J. Med., 1923, xxi, 244–245.
19. Heinecke, H. Ein Fall von multiplen Knochencysten. Beitr. z. klin. Chir., 1903, xl, 481–498.
20. Hirschberg. Quoted by Bloodgood. Loc. cit. ref. 4.
21. Landon, L. H. Ostitis fibrosa cystica. Ann. Surg., 1914, lx, 570–582.
22. Lotsch, F. Quoted by Meyerding. loc. cit.
23. Meyerding, H. W. Cystic and fibrocystic disease of the long bones. Am. J. Orthop. Surg., 1918, xvi, 254–276; 367–382.
24. Muller, G. P. Bone cysts. Univ. Pennsylvania. M. Bull., 1906–1907, xix, 173–177.
25. Murphy, J. B. Osteitis fibrosa cysticas of the upper end of the femur, not involving the head and neck, transplant placed in cavity. Surg. Clin., Chicago, 1913, ii, 783–805.
26. Idem. Tumor of femur; cavity filled with Moorhof wax. Surg. Clin., Chicago, 1913, v, 885–893.
27. Percy, J. F. Osteitis fibrosa cystica; report of a case. Surg., Gynec. & Obst., 1913, xvii, 536–540.
28. Potts, H. A., and Hatton, E. H. Osteitis fibrosa; report of a case. J. Am. M. Ass., 1923, lxxi, 2015–2017.
29. Platou, E. On osseous cysts and so-called giant-celled sarcoma. Ann. Surg., 1918, lxvii, 312–330.
30. Von Recklinghausen, D. Die fibrose oder deformirende, Osteitis, die Osteomalacie und die osteoplastischen Carcinose, in ihren gegenseitigen Beziehungen. Festschr. d. Ass. Virchow, Berlin, 1891, 1–89.
31. Rindfleisch and Harris, T. Eine melanotische Geschwulst des Knochenmarks. Arch. f. path. Anat., Berlin, 1886 ciii, 344–354.
32. Silver, D. The so-called benign cyst of the bones; report of a simulating hip-joint disease; review of the literature; biliography. Am. J. Orthop. Surg., 1912, ix, 563–588.
33. Virchow, R. Ueber die Bildung von Knochencysten. Berlin: 1876, pp. 369–381.

SYSTEMIC BLASTOMYCOSIS

By CHARLES C. GARR, M.D., F.A.C.S., Lexington, Kentucky
Lexington Clinic

THE blastomycete has been known since 1894 when both Gilchrist (2) and Busse (1) described it. Since then many cases have been reported of local blastomycosis but few of the systemic type. A. M. Stober (3) has made the most comprehensive study of the subject and review of the literature of which I know and reports one cure. A letter from him advised me that there has been no material advance in the treatment.

The following case, referred to an orthopedic surgeon by a general practitioner, beginning with symptoms of anything but grave import, passing through a long and painful course, and ending in death, is reported because of the rarity and increasing frequency, and because it is one of the less than 50 reported cases of systemic blastomycosis.

Mr. W. T., age 45, came to see me on July 21, 1923, complaining of a rheumatic like pain in the metacarpophalangeal joint of the left index finger. Two weeks previously this pain began, followed by redness and swelling over the joint and over the dorsum of the hand. About July 1, 1923, he had a nervous breakdown. He became anæmic and lost 14 pounds in weight. Pain of a dull and boring character in the finger had gradually gotten worse so that he could not sleep at night. He had no other complaints except this finger and general weakness and loss of his accustomed vigor.

The mother and father and one sister are living and in good health. Two brothers died in infancy—cause unknown.

Patient had measles, mumps, whooping cough and chicken pox when a child, inflammatory rheumatism 17 years ago, influenza in 1918, and in 1919 a middle ear infection which was very obstinate and would not yield to local treatment but improved and finally healed under iodide of potassium. (This was reported to me by Dr. J. A. Stucky, the otologist who treated him.) To my mind this has a distinct bearing on the onset of the disease.

There was no history of any venereal trouble. He was married and his wife and two children had always been well. For 25 years he was a distiller of whiskey, coming in contact at all times with yeast fungus and since the Prohibition Act he had handled tobacco which often has a mold on it. He has never lived in a damp or mouldy house.

Physical examination. His height was 5 feet, 9 inches; weight, 136 pounds. He was anæmic in appearance, with temperature 99, pulse 80, respiration 18. Head, neck, chest, and abdomen were negative. The metacarpophalangeal joint (left) was swollen, red, and very tender to pressure, the motion of the finger causing pain. There was no fluctuation. No other joint was involved. There was slight tenderness over the epitrochlear glands but no axillary gland enlargement or tenderness. On the left side of the face was a pustule characteristic of a small infected sebaceous cyst.

The Wassermann test and urinalysis were negative. The white cell count was 9,000, with 72 per cent polymorphonuclear.

My first impression was that this was an arthritis, as in his past history he had a polyarticular arthritis. In the following few days a focus of infection was sought in teeth, tonsils and prostate, but these examinations were pathologically negative. The pain in the finger became worse and fluctuation became evident. Aspiration a few days later gave a thin purulent fluid which produced no growth when cultured for 48 hours.

The X-ray examination of the finger showed destruction of the second metacarpal bone (Fig. 1).

Salicylates and baking were given up to the time operation was decided upon.

On August 22, 1923, an incision was made over the second metacarpal bone. The bone was free from its periosteum and was lifted out without any effort. The site was bathed in purulent fluid with caseous appearing material in it. At this time I thought this evidently a tuberculous infection. Cultures from this, however, produced a growth of blastomyces at room temperature but those in the incubator did not grow with such rapidity. (See report of pathologist and Figures 2 and 3.) The infection on his cheek which had been opened by himself and was still discharging was then cultured with the same laboratory findings. August 24, 1923, the hand was much better. There was a small amount of discharge.

He had had night sweats for the past 3 nights. Aspirin relieved the pain. August 26, the left epitrochlear gland was swollen and red. He could not move over without pain. For the past week he had had pain in the first phalanx of the right great toe.

August 30, under gas anæsthesia the great toe was incised, the first phalanx removed, and the periosteum found filled with thick purulent material. Iodoform packing was used and the left epitrochlear gland drained. (Cultures from each showed blastomyces, Fig. 4.)

September 1, he complained of pain in the right knee. The patella was very sensitive to pressure.

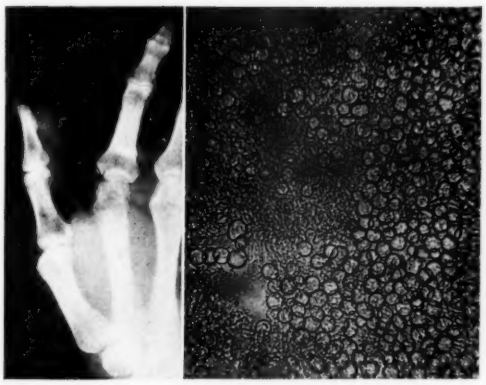


Fig. 1 (left). Blastomycotic destruction of first metacarpal bones.

Fig. 2. Photomicrograph of a primary culture of discharge from the hand.

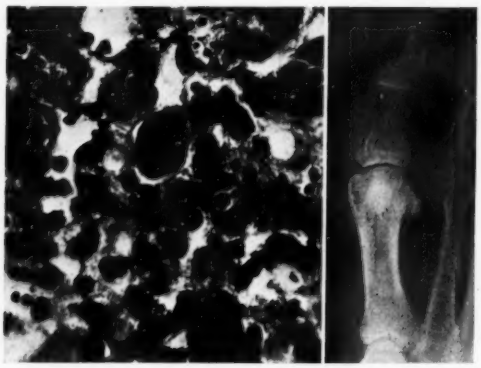


Fig. 3 (left). Photomicrograph of section from clavicle showing budding spores.

Fig. 4. Blastomycosis involving the first phalanx of great toe.

From September to December there was a gradual loss of strength, increasing anæmia, and progressive bone destruction—the left clavicle, the sternum, the right metacarpals, the parietal, and occipital bones all having small abscess formation. There was some improvement in the patella but the pain was never relieved. The inferior maxilla was involved which markedly interfered with mastication and deglutition. Skin manifestations appeared one after another but never healed. These were small areas 1 to 2 centimeters in diameter over the face, scalp, and lower extremities. The chest was examined repeatedly by an internist but always with negative results. There was no cough and scarcely enough sputum to use in laboratory investigations. No blastomycetes, however, were found in the sputum.

October 5, under gas anæsthesia an incision was made to relieve pressure over the clavicle, behind the right ear, and over the sternum. Positive blastomycetic cultures from each abscess were obtained.

There was a gradual increase in the weakness, anæmia, and number of lesions, and complete loss of appetite. It was necessary for him to be kept under opiates at all times on account of pain.

December 24, he became comatose and died December 27.

No post-mortem examination, not even a post-mortem X-ray, was permitted. Repeated examinations of the urine showed only a trace of albumin. An unsuccessful effort was made to culture the organism from the urine. Blood examinations showed a gradual decrease in the hæmoglobin and red blood cells. On October 14 the hæmoglobin was 30 per cent; erythrocytes 3,168,000; leucocytes 8,400; small lymphocytes 15 per cent; polymorphonuclears 85 per cent. Positive blood culture was never obtained though repeatedly tried. The temperature was normal to 100.5 each day with an occasional rise to 101.

TREATMENT

Potassium iodide was given in ascending doses *per orem* but nausea and vomiting contra-indicated more than 130 grains per day. Sodium iodide (30 grains), was given intravenously 5 times causing chill and fever each time. Neo-arsenobenzol (grams .75 was given intravenously 3 times with no apparent improvement.

Local treatment. One per cent copper sulphate solution, iodoform gauze, 1 per cent acriflavine solution, and 2 per cent mercurochrome were successively tried. Acriflavine seemed to keep the wound in a more healthy condition than anything else. Bouillon filtrate of blastomyces with increasing doses was tried subcutaneously but no local or general improvement was noted. A slight local reaction was noted when the larger doses (1 cubic centimeter) were given.

X-ray treatment was tried over the open lesions with apparently no change.

Ordinary diet was given with the addition of gelatinized milk but it was difficult to keep an appetite with the amount of potassium iodide and morphine he was taking.

The laboratory report is the work of Dr. E. S. Maxwell.

Summary of laboratory findings. The first material submitted to the laboratory was pus aspirated from a lesion over the metacarpal bone of the left hand. No bacteria were demonstrated in spreads stained by Gram's method or for acid fast organisms. Cultures produced no apparent growth at 37 degrees C. at the end of 3 days. At this time the cultures were placed at room temperature, and through an oversight were not again observed until the end of 14 days. At this time all inoculated media (nutrient agar, blood agar, Loeffler's blood serum and glucose bouillon) presented colonies. On the solid media the colonies were discrete, dry, light brown in

color, and measured about 1 millimeter in diameter. Microscopic examination showed spherical cells with highly refractile cell walls and slightly granular protoplasm. Many cells were budding. The bouillon media presented round cotton-like tufts that measured about 3 millimeters in diameter. The spores of the organisms isolated were about 20 microns in diameter.

At this time material from the toe and elbow were available for study and, macerated in potassium hydroxide solution, presented typical highly refractile budding spores.

Microscopic examination of sections of tissue from the hand showed a marked inflammatory reaction with necrosis and foreign body giant cells. A mistaken diagnosis of tuberculous inflammation was first made. After the nature of the lesion was recognized from the cultures, a restudy of this tissue with oil immersion objective showed many budding spores similar to that described above. Later blastomyces were demonstrated in tissue from clavicle, skull, and other lesions. In the older lesions many very small spores were noted.

Repeated blood cultures with large quantities of blood and large quantities of media were negative. Blastomyces were never demonstrated in urine or sputum. The organisms grew readily on all ordinary culture media. On the primary cultures a radiating mat of mycelia surrounded each colony at the end of the third week. In a few days aerial hypha appeared and in time filled the culture tube. In the second and third transplants the mycelia appeared earlier, and after the third transplant colonies of spores did not appear. Subcultures grew readily from cultures that have been at room temperature for 12 months, although the aerial hypha are not pronounced in the recent plants.

REFERENCES

1. Busse. Zentralbl. f. Bakteriol., xvi, 175.
2. Gilchrist. Johns Hopkins Hosp. Bull., vii, 129.
3. Stober, A. M. Arch. Int. Med., 1924, xiii.

DEPARTMENT OF TECHNIQUE

COMBINED SATCHEL HANDLE OR TUBED PEDICLE AND LARGE DELAYED WHOLE SKIN PEDICLE FLAPS IN A CASE OF PLASTIC SURGERY OF FACE, NECK, AND CHEST

By B. F. ALDEN, M.D., F.A.C.S., San Francisco, California

THE object of this paper is to present a case of severe second and third degree burns with resulting cicatrization and contracture of tissues of face, neck, and thorax, and to describe a series of operations to correct the defect, and the end-result after 3 years following the extensive and complicated adaptation of the satchel handle and delayed pedicle graft method which was used and described during the World War by Gillies and Blair and later by G. B. New of the Mayo Clinic, C. C. Coleman, and probably others.

B. L. U., a female, age 5 years, a native of Kansas, a healthy normal child previous to the time of the accident, July 4, 1921, was severely burned about the head, face, the anterior and lateral cervical region, and the anterior surface of the chest as far down as the level of the umbilicus.

She was brought to the writer 3 months later, at which time the chin was fused to the sternum by firm cicatrization, completely obliterating the anterolateral cervical contour, as illustrated in Figures 1 and 2. A deep unhealed ulcer, with cicatrized leathery edges about 5 by 8 inches over the anterior aspect of the chest and sternum, was closed by Thiersch grafts at the end of the fourth month after the injury.

On January 26, 1922, the first of a series of plastic operations was begun. The technique is described and the results are illustrated by a series of photographs taken during the progress of the case.

First operation. January 26, 1922. Ether anæsthesia was used. Two tubed pedicles 8 inches in length were formed from the integument over either scapula, as illustrated in Figures 2, 3, and 4. Healing was rapid, so that by the end of the third week the area could be sufficiently sterilized for further surgical procedure. Figures 3 and 4 illustrate the condition 3 weeks after the first operation.

Second operation. February 16, 1922. The integumentary flaps, each 4 by 5 inches in area, on either side of dorsum were cut out, leaving the lower end of the tube pedicle attached to the superior medial aspect. The flaps were lifted from the surrounding tissues, replaced, and retained in the original bed, by interrupted silkworm-gut and dermal sutures.

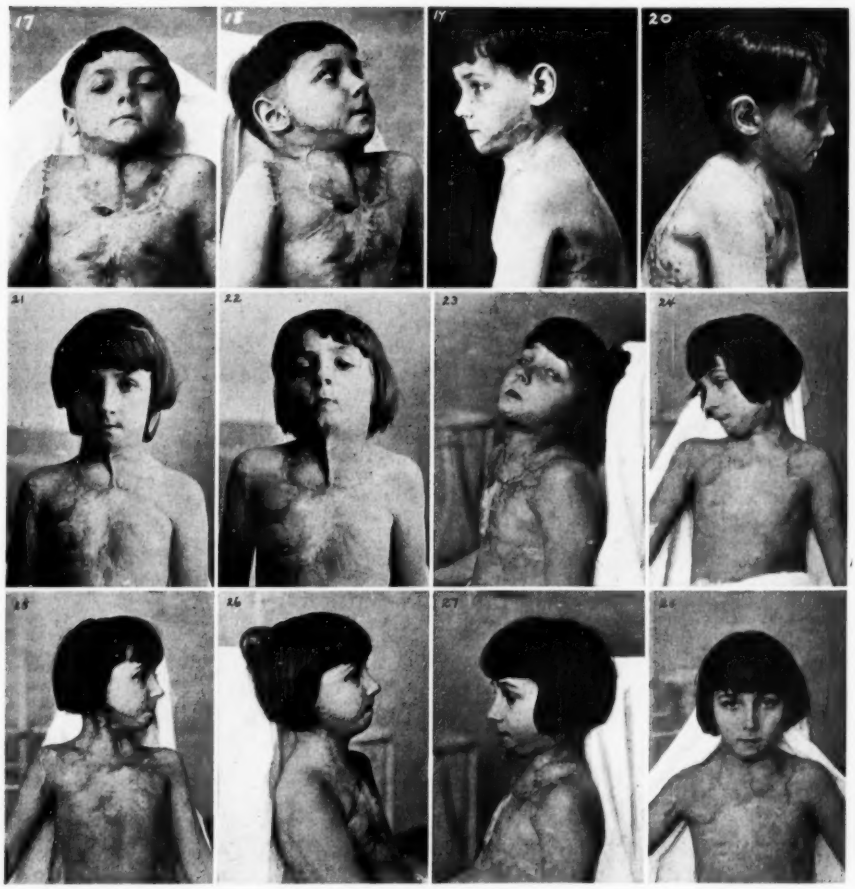
Figure 5 presents the condition of the delayed pedicle graft flaps 10 days after the second operation, just prior to the transfer of the flaps to the anterior cervical region.

Third operation. March 6, 1922. The scar tissue was thoroughly removed from the anterior and lateral aspect of the lower jaw, the anterior and lateral cervical region, and the anterior chest wall, to a line ½ inch below the lower margin of the clavicle, disembedding both delayed pedicle flaps, swinging them over either shoulder and suturing them into place in the defect in the neck. The posterior faults resulting from removal of the delayed pedicle flaps were readily closed by undermined, sliding flaps, and they healed readily. After the removal of all scar tissue, including the cicatrized and shriveled remnants of the platysma myoides, when the head was thrown back and the chin lifted, a gaping defect 5 by 8 inches presented. The delayed graft flaps were sutured above to the integumentary line of the neck, face, jaw, and chin; below, to scar tissue and integument along the inferior border of the clavicle; laterally they were fitted in and sutured to the lateral cervical integumentary line and joined together in a median line by interrupted dermal sutures. To fit in, adapt, and hold the flaps to conform to the contour of the neck and to obliterate the dead spaces, loosely tied mattress sutures were sparingly used. A compress of gauze, moistened with a weak solution of mercurochrome was now applied and held in place with moderate pressure. Serum continued to accumulate in pockets beneath flaps for several days. These areas were evacuated as they formed and drained for a few hours with twisted silkworm gut. Within 1 week the flap and wall adhered throughout, presenting the appearance, 8 days after operation, shown in Figures 6, 7, 8, and 9. No sloughing occurred even at the periphery of the flaps. The dark appearance presented in the photographs is largely due to the deep staining of scarf skin from mercurochrome solution used on compresses.

Fourth operation. April 8, 1922. At this time it was found that more vertical shrinkage had occurred in the flap than in the horizontal diameter, resulting in considerable down pull on the chin. This was especially noticeable in the median vertical scar line of the flaps. The upper or proximal end of the tube pedicle was detached and split two-thirds of the way along the line of the old scar, toward the delayed graft pedicle attachments. The scar line of the lower margin of the mandible from the symphysis to the angle of the jaw was removed, gaps were widened by careful dissection, and the split grafts inlaid and sutured in place. The appearance after healing is shown in Figures 10, 11, 12, 13, 14, 15, and 16. Rapid primary union resulted throughout from this procedure, and after an interval of 17 days the patient was prepared for the fifth operation.

Fifth operation. April 25, 1922. The remaining, or distal attachment of tubed pedicles were detached, split, and the same procedure enacted for the posterior upper margin of the defect as described in the fourth operation, with the result in 2 weeks shown in Figures 17 and 18, and later after complete healing in Figures 19 and 20.

Figs. 1 and 2. Photographs showing extent of scar at time patient was brought to hospital.
Figs. 3 and 4. Condition 3 weeks after the first operation.
Fig. 5. Condition of the delayed pedicle graft flaps 10 days after the second operation.
Figs. 6 to 9. Condition 8 days after operation.
Figs. 10 to 16. Condition after healing from fourth operation.



Figs. 17 and 18. Condition 2 weeks after fifth operation.
Figs. 19 and 20. Condition later after healing, fifth operation.
Figs. 21 and 22. Condition in October, 1923, showing considerable keloid formation and contracture of the mid-line scar from chin to sternum.
Figs. 23 to 28. The scars have been excised and the edges of the flaps zigzagged to obtain length at the expense of the lateral diameter.

The patient was then permitted to return to her home in Kansas with instructions to return after an interval of a year or a year and a half for correction of any contractures which might occur during the interim. In October, 1923, the child was again brought under observation. At that time she showed considerable keloid formation and contracture of the mid-line scar from the chin to the sternum, giving the center board appearance shown in Figures 21 and 22. To a lesser degree the same condition obtained in the vertical scars on either side of the neck. These scars were excised and the edges of the flaps zigzagged in order to obtain length at the expense of the lateral diameter. Figures 23, 24, 25, 26, 27, and 28 illustrate how perfectly this object was accomplished. The patient has complete functional movement of the head, chin, and neck, the normal contour and poise of the head and chin are re-established, the skin flaps have remained soft and pliable, the color has become almost the same as the skin of the face and neck, there has resulted no œdema of any areas in or adjacent to implantation field, and sensory nerve function to a considerable degree is re-established over the area engrafted. To accomplish this result there were in all 72 square inches of full thickness skin transplanted from the scapular region to front of the neck without the loss of any appreciable amount of integumental tissue; 20 square inches in each delayed flap, or 40 square inches, 16 square inches in each tubed pedicle, or 32 square inches. The proximal ends of tube pedicles were placed in the most advantageous position for shifting flaps, without regard, as has been our usual custom, to the vascular supply.

Fig. 23a. Zigzag of lateral scars to overcome contractures. Stains from mercurochrome dressings.

Fig. 23b (right). Final operation. Zigzag lateral scars. Showing result of previous zigzag of median contraction cicatrix.

The delayed flap assures pre-knowledge of viability of grafts before the operation for transfer is undertaken. The grafts become to a certain extent fixed, and while they are flexible and easily adapted to any surface, they shrink much less than do recently dissected flaps. The presence of newly formed granulations and capillary blood vessels on the under surface of delayed flaps assure a more rapid union with the freshly denuded surface upon which they are engrafted.

In this case we were especially fortunate in that no mucous surface presented to be covered, nor was prosthetic appliance necessary to establish plastic contour.

To mention a few drawbacks met with in cosmetic and plastic surgery of this nature, by far the most important to the surgeon is the tendency to exuberant cicatrization or keloid formation along or adjacent to the suture lines, when subjected to undue tension. Pulling on the scar, from whatever cause, must necessarily be prevented or contracture and deformity will surely result. The method of approximation of subcutaneous edges by eversion, as described in 1923 by Dorance and Bransfield, is a long stride in the right direction for the prevention of exuberant scar formation. Overgrowth of hair on surface of grafts, as well as the slight difference in texture and color, are minor objections to the surgeon, but loom large in the mind of the female patient, and therefore demand due consideration to the end that such defects may be overcome.

THREE-STAGE OPERATION FOR RADICAL RESECTION OF THE COLON FOR CANCER

With Report of a Case

By JAMES G. MONTGOMERY, M.D., F.A.C.S., Kansas City, Missouri

THE following case is reported for the reason that it presents four very interesting features; first, a differential diagnosis of tumors of the colon, because the filling defects are characteristic of an intraluminal growth; second, the unusual occurrence of an intussusception of the carcinomatous cæcum and ascending colon into the transverse colon; third, the preparation of an apparently inoperable case to withstand a surgical operation, and fourth, the radical resection of the colon under local anæsthesia with excellent results.

Mrs. M., age 72 years, began to give marked symptoms in May, 1919, characterized by loss of weight and constipation, alternating with pain in the abdomen as if she had taken a severe cathartic, followed by diarrhœa and passage of blood and mucus. An epigastric mass was first observed in October, 1919. It seemed to vary in size with passage of gas and diarrhœa. Her normal weight was 130 pounds, but in October, 1919, she weighed only 79 pounds. She ate very little and was annoyed by more or less constant pain in the abdomen.

Physical examination. The patient is anæmic, emaciated, weak, walks with difficulty and is confined to bed most of the time. Her general physical condition is without interest except for the suggestion of malignancy. The abdomen presents a transverse fullness above the umbilicus that is sausage shaped and measures 5 by 3.5 inches in greatest dimensions. It is hard and cartilaginous at its distal end, soft and doughy proximally, and has a wide range of mobility especially in the right abdomen. Intestinal patterns are present and there is hyperperistalsis and general tympany. The blood count shows hæmoglobin, 35 per cent; red blood cells, 2,320,000; and white blood cells, 9,000.

Diagnosis. The presence of blood and mucus in the stools with tenesmus, increased peristalsis and tympany indicates a tumor of the large gut. The presence of the tumor above the umbilicus suggests that it is located in the transverse colon. This supposition is verified by the mobility of the mass and by the termination of the bismuth enema at the distal extremity of this tumor mass (Fig. 1). The harder distal extremity, in all probability, is the tumor mass itself, while the softer more compressible proximal portion a fæcal mass retained by the tumor.

Treatment. The presence of mechanical obstruction obviously indicates the operative removal of the mass or colostomy. Her general condition is such, however, that the likelihood of her surviving an operation is small. Operation, therefore, is contingent on the possibility of building up her general condition to a point where operation will not be unduly hazardous.

Pre-operative treatment. She was given a high caloric diet every 2 hours from 7 a. m. to 10 p. m. The diet consisted mainly of milk and cream, eggnogs, egg albumens, creamed custards, egg malted milk, etc. Two days preceding the operation, water was forced by mouth; she was given ½ dram of soda in ½ glass of water every 2 hours from 7 a. m. to 9 p. m. and was also required to eat as much as possible of a pound of glucose stick candy. Her irritable bowel prevented the use of a 24 to 48 hour pre-operative proctoclysis of glucose and soda. Her assimilation was so poor and anorexia so extreme, in spite of the forced feeding, that she made no gain. Transfusion was then resorted to and her blood count indicates the rate of improvement:

October 7, 1920. Hæmoglobin was 35 per cent, red blood cells 2,320,00 and white blood cells 9,000.

October 8, 1920. She received 600 cubic centimeters of blood by the Lindeman method.

Fig. 1. The filling defect produced around the ileocæcal valve tumor mass in the transverse colon, by giving a bismuth enema.

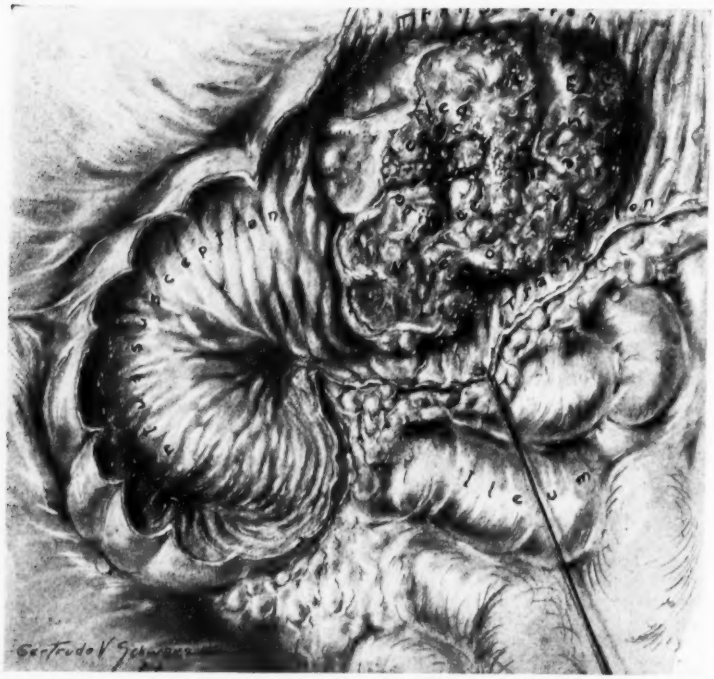


Fig. 2. The ileocæcal valve and surrounding cauliflower growth in the transverse colon, also the area of intussusception.

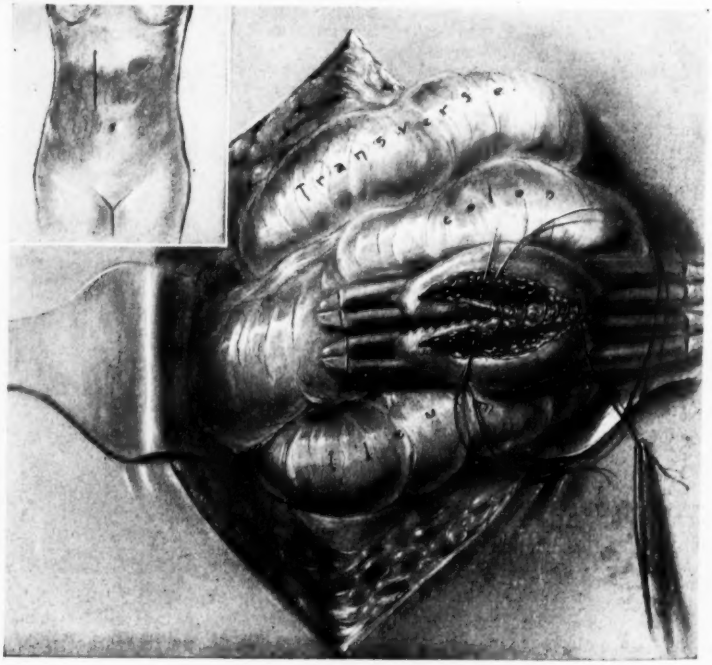


Fig. 3. The location of the skin incision and enterostomy between the terminal ileum and proximal to the splenic flexure of the transverse colon.

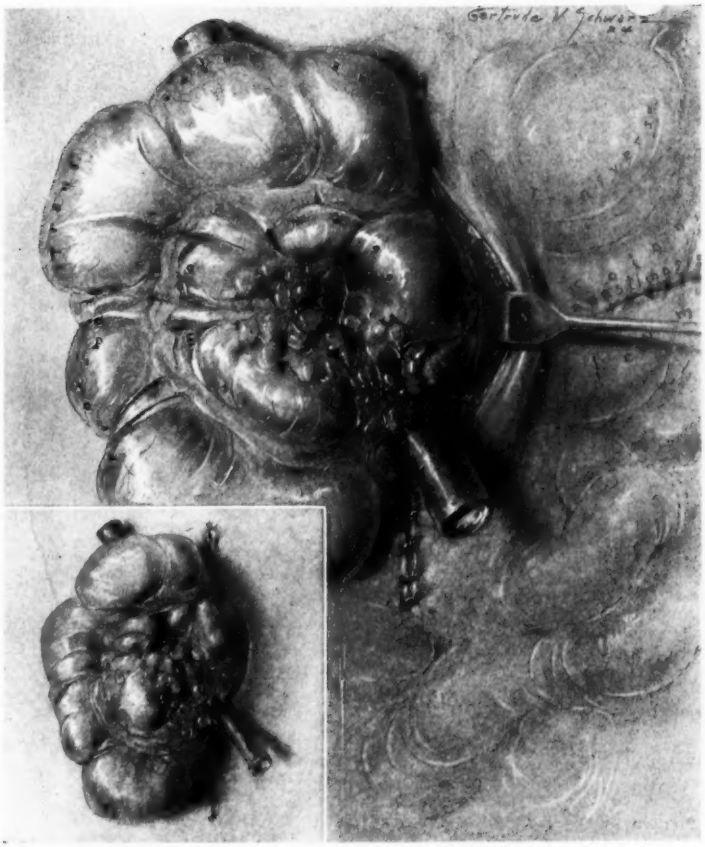


Fig. 4. The mass retained by a rubber tube through the mesocolon, the colon anchored to the ileum under the mass and in shadow the enterostomy. The insert shows wound closure around the tumor.

October 9, 1920. Hæmoglobin was 55 per cent, red blood cells 3,500,000 and white blood cells 9,000.

October 10, 1920. She received 650 cubic centimeters of blood by the Lindeman method.

October 11, 1920. Hæmoglobin was 60 per cent, red blood cells 4,000,000, and white blood cells 8,000.

When this state was reached, operation seemed justified. October 12, 1920, the first stage of the operation was performed. In order to preserve the patient's physical resources as much as possible, local anæsthesia (½ per cent novocain and adrenalin) was elected, preceded by a hypodermic of morphine. A low epigastric incision was made a little to the right of the mid-line. The abdomen was opened and the mass was found to be an intussusception of a short piece of the ileum, the entire cæcum, and ascending colon into the transverse colon (Fig. 2). The mesocolon was long enough to permit the mass to be delivered upon the abdomen. The mesenteric glands and liver were free from evidence of metastasis. A lateral anastomosis was made between the terminal ileum and transverse colon proximal to the splenic flexure by a continuous linen suture in the serosa and a Connell inverting suture of plain catgut. The gastro-enterostomy clamps were used to maintain approximation and control bleeding during the suturing (Fig. 3). The growth was then delivered upon the abdomen, sliding a 1 inch rubber tubing through the mesocolon under the loop of colonic mass. The ileum was sutured to the colon under the mass and both to the peritoneum by interrupted plain gut sutures (Fig. 4). The wound was then closed as tightly as possible without interfering with the circulation of the tumor mass. The latter was covered with strips of sterile vaseline gauze and its moisture and warmth maintained by warm saline packs which were changed every 4 hours for 6 days. The circulation during this time remained good and the wound completely walled off with no drainage.

The patient began passing gas and some fæcal material, October 13, 1920, the day following the operation, showing that the anastomosis was patent. Her abdominal pain stopped immediately and there was less than usual postoperative tympanites. Her maximum postoperative reaction was: temperature 101 degrees, pulse 120 and respiration 24. She was given 600 cubic centimeter of normal saline by hypodermoclysis.

October 14, 1920, the maximum pulse was 108, temperature 100 degrees and respiration 20. Liquid nourishment

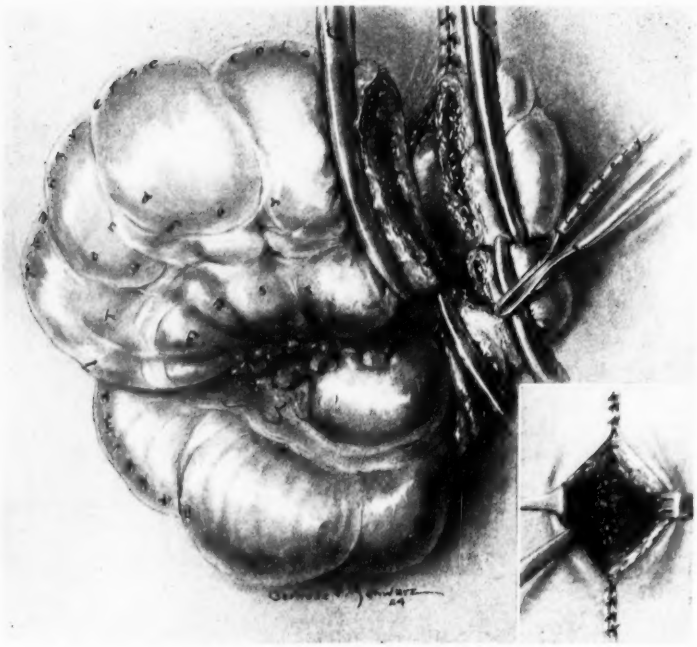


Fig. 5. The amputation of the tumor mass. The insert shows the inverted stumps in final closure.

was given every 2 hours. October 15 and 16, 1920, her temperature was normal.

October 17, 1920, 6 days after the primary operation, the second stage was performed. The ileum and colon were clamped off with large crushing hæmostats and the mass cut off with an electric cautery. The stumps of ileum and colon were inverted with pursestrings of catgut and then sutured together (Fig. 5). No anæsthetic was employed for this stage, but morphia ¼ grain should have been given as the patient had considerable distress in the back.

October 28, 1920, 16 days after the operation, the patient was walking for the first time in 2 months.

November 10, 1920, 1 month after the operation, her blood count was as follows: hæmoglobin 70 per cent, red blood cells 4,320,000. Her gain in weight was 13 pounds.

Both ends of the gut drained following the amputation of the mass, most of the fæcal material coming from the colon. The lumens gradually diminished in size until she would go a day or so at a time with no drainage at all, then she would have a spontaneous discharge of fluid fæcal material.

December 10, 1920, at this point in her progress, she was dismissed from the hospital.

January 18, 1921, she reported a gain in weight of 30 pounds.

August 2, 1921, she returned to the hospital with two very small fistulous tracts still draining intermittently. Her general condition had approximated normal. There was no evidence of metastasis and the X-ray showed the ileocolonic anastomosis functioning properly. It was then thought advisable to close the fistulæ.

Technique of the third stage of the operation. Morphine ¼ grain and atropine 1/150 grain were given hypodermically and followed by gas oxygen.

The skin was incised around the wound and the fistulæ approached from all sides as in a

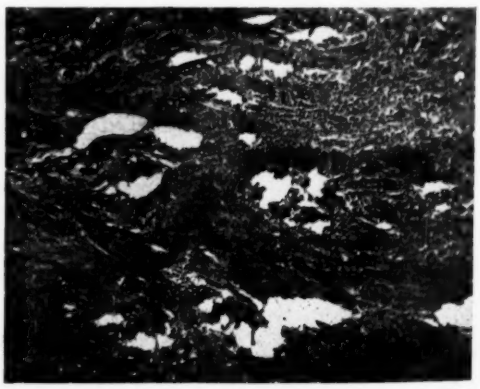


Fig. 6. Photomicrograph of the tumor showing the dense medullary type carcinoma.

ventral hernia, the fistulæ and infected field being isolated by hæmostats on the skin margin. Laparotomy tapes were used to protect the outer skin margin. The two fistulæ were close enough together so that the final stump was clamped in a hæmostat as an appendicular stump, cut off, carbolized, ligated, and inverted with a double chromic gut purse string. The marginal wound adhesions were liberated and the usual closure was made with catgut in the peritoneum, chromic No. 2 in the fascia, silk worm retention sutures and the skin with silk. A strip rubber dam drain was inserted in the lower angle of the wound, and after this was done a moist hot dressing was applied.

August 9, 1921, 7 days after the third stage of the operation, she had some serous drainage which grew bacillus coli on culture.

August 18, 1921, she went home in good condition, with wound closed.

January 1922, 1 year and 4 months after the operation, the patient reports that she feels quite well and is doing her own house work.

Pathological report. The specimen is composed of the cæcum in which the growth is located, 4 inches of the ileum, the ascending colon and 6 inches of the transverse colon and adherent omentum weighing 590 grams. The growth involves most of the cæcum and all but ¼ inch of the tip of the appendix. The cæcum is inverted and lies in the proximal portion of the transverse colon. The ascending colon (Fig. 2) is haustrated like an accordion bellows by traction of the growth in the direction of the fæcal stream. The neoplasm measures 3½ by 3 inches in greatest dimension, is friable, irregular, looks like a cauliflower and has a stenosed ragged ileocæcal opening through which the fæcal stream emptied into the transverse colon. The portion of the transverse colon forming the bed of the growth is thinned; the serous surface is injected and the omentum is adherent. The terminal portion of the ileum is not involved by the growth.

Microscopic section shows a dense medullary type carcinoma (Fig. 6).

The pathological diagnosis was carcinoma of the cæcum with intussusception of the cæcum and ascending colon into the transverse colon.

CÆSAREAN SECTION AFTER THE TEST OF LABOR

Description of Author's Technique

By H. M. ARMITAGE, M.D., F.A.C.S., Chester, Pennsylvania
Surgeon, Chester Hospital

THE method of performing cæsarean section depends upon the stage of labor and the complications present in the individual case.

When the operation can be performed before labor begins and the woman is free from infection, a short, high incision with the uterus opened in the abdomen and hooked out with the forefinger will probably remain the operation of choice with the majority of surgeons. This operation has been termed the "classical cæsarean section" but it offers two distinct disadvantages in contaminated cases.

1. The "spill" enters the peritoneal cavity at the time the operation is performed.

2. The infected lochia may seep into the uterus wound and into the general peritoneal cavity after the operation has been performed.

The mortality rate should be very low with the classical operation under ideal conditions but ideal conditions are often absent and the mortality rate is not as low as we are led to believe in a study of statistics from some of the well organized clinics throughout the country.

E. Holland (5) reported 3,314 cæsarean sections performed throughout Great Britain from the years 1911 to 1920 in which the average mortality was 4 per cent; with a mortality of only 1.6 per cent in the early uncontaminated cases, but 27 per cent in the late cases or in those in which delivery had been attempted. E. L. King (7) reported 117 abdominal cæsarean sections at the Charity Hospital of New Orleans (excluding the Porro cases), in which there were 12 deaths from peritonitis, and 2 from sepsis; all these patients had vaginal examinations or attempts at delivery before operation. Most of the 76 patients recovering had fever, the puerperium being absolutely afebrile in only 17.

In studying the cause of death, infection stands out as the predominating factor. The atria of invasion occurs in exactly one place, the vagina, and while it is well known that the infection is in direct proportion to the length of time a woman has been in labor, rupture of the membranes, the number of vaginal examinations, and the attempts at delivery either manual or instrumental, there still remain many women who are sent into the hospital for cæsarean section after these principles have been violated.

If it is true that most puerperal uteri contain pathogenic organisms by the fifth or sixth day, the logical deduction would be that the suture line offers the most favorable area for invasion of the peritoneal cavity. Polak (9) gives the incidence of pathogenic organisms in the uterus as 28.8 per cent in uncontaminated cases and 50 per cent in cases in which the patient has been previously examined.

TYPES OF OPERATION PERFORMED IN CONTAMINATED CASES

In order to avoid the unjustified risk to the mother which is inevitable when the classical section is used in contaminated cases, several other forms of abdominal delivery have been introduced. The extraperitoneal methods which have for their object the avoidance of opening the peritoneal cavity, were largely developed in Europe, because so many women are referred to the continental clinics by midwives after many examinations. Joerg, as early as 1809, performed the extraperitoneal operation by gaining access through the flank. He was followed by Selheim who pushed the peritoneum from the bladder anteriorly, and later by Latzko, who preferred to separate the peritoneum upward off the lower uterine segment with the bladder forced to one side.

In 1908, Frank (4) of Cologne, operated in contaminated cases by means of a transverse incision with enough separation of the peritoneum from the bladder and the anterior surface of the uterus for delivery of the child, the parietal peritoneum first having been united to the uterine peritoneum in order to shut off the peritoneal cavity. Doederlein approached the uterus through an incision parallel to Poupart's ligament. The true extraperitoneal operation has never been widely adopted in this country because of the technical difficulties encountered and the liability of injuring the bladder or ureter.

Kroenig (8) advanced the thought that the superior results in contaminated cases were not due to the fact that the operation was performed in an extraperitoneal manner but because the incision was made in the thin non-contractile lower portion of the uterus and hence the wound could heal in peace and not be subjected to the tugging

and pulling due to the contractions in the thick body of the uterus. This muscle grinding actually pulls apart the edges of the incision and allows infected blood to fill the gaps. The danger of rupture of the incision during after-pains is so great that Holland in his studies came to the conclusion that the rupture incidence was two and a half times greater when catgut was used than when silk was used and that silkworm gut was the best of all. Many operators using catgut insist upon the interrupted stitch, feeling that the continuous stitch, when used alone, does not adequately take care of the relaxation due to the unrest of the uterus. Many of the deaths ascribed to surgical shock are probably due to rupture of the uterine incision.

Kroenig devised an operation which opened the peritoneal cavity by a low longitudinal incision, separated the bladder from the uterus, made a longitudinal incision in the lower part of the uterus, emptied the uterus, sutured the uterine incision, and covered the incision with bladder by suturing the bladder peritoneum above the upper aspect of the uterine incision.

Beck's (6) technique differs from the Kroenig operation in only one respect. An upper flap of peritoneum is raised after the bladder is separated. After the uterine incision is closed the upper flap is sutured down over the upper part of the incision and the lower flap is brought up over this in such a fashion that a double layer of peritoneum safeguards the uterine incision.

De Lee (2) has further modified the operation by uniting the fascia between the bladder and the uterus as a separate layer and using a suction apparatus to empty the uterus. J. C. Hirst and Van Dolsen adhere to the Beck technique with the modification of a gauze pack across the lower part of the abdominal cavity for protection against the infected "spill."

DISADVANTAGES OF LOW OPERATION

The low operation as ordinarily performed is more difficult technically than the classical operation and requires a longer time.

The patient must be well advanced in labor; but she generally is, or the surgeon would hardly consider this type of operation. With the exception of the Hirst and Van Dolsen operation where gauze is used as a pack, no adequate protection has been given to the general peritoneal cavity against the infected "spill," although the operation adequately safeguards against infection spreading through the incision.

The opponents to the low operation have based their arguments upon the fact that during the operation infected blood and liquor amnii find their way into the peritoneal cavity with all the methods thus far devised.

Paddock, Heaney, and Holmes believe that there is as much danger of the "spill" getting into the peritoneal cavity in the low operation as in the classical operation. Paddock substantiates his assertions by statistics and personal observations.

The opponents also quote Munro Kerr as saying 25 years ago that the one advantage of the Fritsch incision was that it was farthest removed from the potentially infected cervix.

METHODS DEVISED FOR OVERCOMING DISADVANTAGES

Those who are enthusiastic over the advantages of the low operation quote Munro Kerr (6) as saying at a later period in his life that healing in the cervix is better than in the fundus because active involution and fatty degeneration of the uterine wall defeat the healing power of the tissues.

The methods devised for protecting the peritoneal cavity during the operation are walling off with gauze, attaching the visceral to the parietal peritoneum, the suction pump, and the method about to be described.

The operation in which gauze is used as a coffer dam has been well described by J. Hirst and Van Dolsen and their results have been excellent in an extended series of cases.

When the visceral and parietal peritoneum are sutured together the uterus often pulls away from the abdominal wall, especially when a large child is encountered, and the stitches or forceps are pulled out, the purpose of the operation thus being defeated. Newel has stressed the fact that the suture line was not infection proof. My personal belief is that unfortunately one secures apposition of suture lines rather than an effective barrier against the spread of infection.

The suction pump does not remove all of the infected liquor amnii and when the child is delivered a gush of infected fluid comes with it.

All these methods have given good results as in fact have the methods in which no precautions have been taken to protect the peritoneal cavity; but all the important statistics on the results of the low operation have emanated from the large obstetrical clinics where refinement of individual skill and the adoption of ultramodern principles of surgery influence, to a great degree, the low mortality rate.

De Lee and Cornell (3) flatly state that the mortality rate of cæsarean section is high because

the operations are performed by general surgeons in most instances. It is absolutely necessary, therefore, that any operation which is to be widely adopted should not only be one which the general surgeon may readily perform but one which will afford effectual protection of the peritoneal cavity at the time of operation by the less experienced man.

Before describing the operation which we have adopted to fulfill these requirements I wish to state that absolutely no claim to originality is made and the following technique is simply a detailed description of the various steps which I have found to be easy and safe when a general surgeon must attempt these operations.

TECHNIQUE

The lower uterine segment should be well distended since if this has not occurred the uterine incision can not be made without extending the cut into the body of the uterus. In other words the patient should have been in labor a few hours so that the cervix is some distance away from the bladder. A midline incision from the umbilicus or a little above to the symphysis pubis is made. The peritoneum is opened. The uterus is eventrated out of the incision and the abdominal wall closed with clamps or silkworm gut sutures close up to the uterus so that it is forced down toward the symphysis. Several large flat sponges are then spread out over the abdominal wall under and to the sides of the uterus or if the incision has not been closed tightly, rubber dam is used under the sponges. Two sponges are placed in the lower angle of the incision on each side.

It will be noted that up until this time the hand of the operator has not become infected by contact with the contents of the uterus and no harm can occur from introducing the sterile gloved hand into the peritoneal cavity. A transverse incision is made in the peritoneum of the uterus about a half inch above the junction of the uterus with the bladder. The bladder with the lower peritoneal flap is dissected below and is pushed down well toward the vagina. We next make a longitudinal incision in the center through the muscular layers of the uterus. It is probably better to start the incision from below so that blood will not obliterate the field.

The membranes are ruptured, the face is rotated anteriorly, and the head is delivered with forceps, if it is impossible to deliver it with the hand. I usually am able to deliver it with the hand.

The hands and instruments are now infected for the first time with the infected contents of the uterus. The uterus is crowded down hard against the symphysis pubis over which most of the drainage flows. Pituitrin is injected into the arm, not the body of the uterus. If the placenta does not separate spontaneously it is gently removed from the uterus, care being taken to remove all the membranes. If the placenta is hurriedly separated hæmorrhage is apt to occur. The uterine wound is then sewed up with No. 2 twenty-day catgut, interrupted sutures being used for the first layer, the uterine muscle being embraced down to but not through the endometrium.

The second layer is a continuous No. 2 twenty-day catgut suture which catches the uterine muscle. In the upper part of the wound where the muscle is thick enough, we place the interrupted stitches through the lower third of the uterine wall and the continuous stitch through the upper two-thirds of the wall. In the lower part of the incision the wall is so thin that we can not suture in layers and we catch the uterine muscle outside of the interrupted stitches and bring it over the first row of sutures. The fascia between the bladder and uterus can be seen as a glistening layer and is always brought over the first row of stitches by means of the second line of sutures, the needle biting into the fascia and muscle with each suture. We have never sewed the fascia as a separate layer, feeling that stronger union is attained by the above method.

We formerly used interrupted sutures for both layers as we always do in the classical cæsarean, but now use one layer of continuous sutures because the lower segment is at rest and is not apt to snap the continuous suture as could readily occur with the tugging and grinding which occurs during healing in the body of the uterus. If the incision is carried up into the body of the uterus on account of a large child, great care should be used to place the interrupted sutures close together because the motion in the upper uterine segment is different from that in the lower and a seesawing movement is set up which may actually pull the edges apart if reliance is placed upon a continuous suture alone.

Another point to be considered by one unaccustomed to this operation is that it is easy to overlook an opening in the lower angle of the incision after one has apparently sutured the entire wound. We overcome this by not cutting the lower interrupted stitches and pulling the incision well out from under the bladder in order that the wound may be inspected.

The upper peritoneal flap, if there is one, is now tacked down over the upper part of the incision by a few interrupted catgut stitches. The lower peritoneal flap is brought up over the upper

limits of the longitudinal incision and stitched with a continuous suture of catgut. The uterus which has been covered with a warm sponge is now cleansed with sponges wet with warm saline solution, the pads behind the uterus are removed, and the abdominal wall cleansed. The operator and assistant now leave the table while a second assistant, who has up to this time not assisted at the operation, removes the clamps and replaces the uterus in the abdomen. Clean towels are placed about the incision. *The surgeon and nurse change their gloves and gowns. A fresh set of instruments are then used to close the abdominal incision, with the second assistant acting as first assistant.* By this method the "spill" is prevented from entering the peritoneal cavity in an effectual manner, there is no danger of the peritoneal cavity becoming infected from either dirty gloves or instruments, and the operation is easy to perform.

RESULTS OF OPERATION

The number of cæsarean sections that I have performed totals 52 at the present time. These represent unselected cases, in a large number of which there had been interference before operation. In the early cases attempts were usually made to deliver the child with instruments, and when it was impossible, a cæsarean was decided upon. The classical cæsarean was performed until 1920 but it was felt that the risk was too great in the class of cases upon which we were called to operate. There were 6 maternal deaths in the whole series, 5 prior to 1920 and 1 death in 31 operations since 1920, when the technique which has been described was first adopted. The 1 woman who died had had many vaginal examinations, and ruptured membranes, the uterus and contents were infected so badly that a hysterectomy was performed, and she died in the third week from blood infection.

Of the 5 deaths before 1920, 2 were from peritonitis (postmortem examinations were held in both cases), 1 was from hæmorrhage from the uterus on the fourteenth day (postmortem examination revealed no peritonitis, a healed uterine wound, and a uterus filled with débris), 1 from eclampsia 2 weeks after operation, and 1 patient died on the table as the skin sutures were being tied. The last death was ascribed to pulmonary embolism although there was no postmortem examination.

Two deaths in 21 cases were directly attributed to peritonitis. Thus it will be seen that, in my hands at least, the classical cæsarean carried a very grave maternal risk in infected or potentially infected cases.

The low operation, performed as described, has evidently lowered the mortality, although too few operations have been performed to positively prove this assertion.

I am quite convinced that the technique and not a more careful selection of cases during the last few years has been responsible for the lower death rate in the 31 cases since 1920.

The following cases will at once show the serious types upon which we have been in the habit of performing the operation outlined.

CASE REPORTS

The operations in these cases were all performed by H. M. Armitage and F. R. Nothnagle.

A. G., 1921, had been in labor for 3 days. The membranes were ruptured and many vaginal examinations had been made. A low cæsarean operation by the author's technique was done. There was some slight infection of the wound but no symptom of general infection. The patient recovered.

H. M., 1921, with rachitic pelvis, had been in labor 24 hours. Forceps had been used. She entered the hospital with a temperature of 100 degrees F. A low cæsarean by the author's technique was done. The temperature was febrile for about a week. Patient recovered.

H. E., 1922, was in labor 19 hours. The membranes were ruptured and many vaginal examinations had been made before she entered the hospital. She had had 1 child 16 years previously, the delivery being very difficult. The head was not engaged. A low cæsarean section was done, the author's technique being used. The temperature was 100 degrees F. for 2 days following operation and then became normal. There was no infection of the wound. Dilatation of the stomach followed operation, and the stomach was washed out. Recovery followed.

A. W., 1922. This was a breech presentation, with the feet hanging out of the vulva. Two physicians had tried to effect delivery but found it impossible. The patient was very fat, weighing 240 pounds. A low cæsarean by the author's technique was done and a living child delivered. Stormy convalescence with great abdominal distention, but no rigidity, followed. The distention was relieved by a stomach tube. Recovery followed.

H. K., 1924, a dwarf, 16 years of age, was in labor 2 days. The membranes ruptured, 24 hours. The pelvis was contracted. A low cæsarean by the author's technique was performed. The lochia was very foul for several days after operation. There was no infection of the incision, and the patient recovered.

M. F., 1924, was in labor 36 hours. The membranes ruptured, 12 hours. One examination had been made outside the hospital. The pelvis was contracted. A low cæsarean was performed by the author's technique. Slight infection of the wound followed. The patient left the hospital in 16 days.

R. H., was in labor 4 days. Pains were so severe and frequent, 48 hours before admission, that the attending physician remained at the home of the patient all night. The nurse stated that pains had occurred every 3 to 5 minutes the night before operation. The patient was very weak. The membranes had not ruptured. Many vaginal examinations had been made before admission.

A low cæsarean operation by the author's technique was performed, and a living child delivered. The temperature

was 102.3 degrees F. the night of operation, and 104.2 on the second day. It dropped on the seventh day to normal but every evening went to 100 until the fourteenth day. Good recovery followed.

SUMMARY

1. The low operation is indicated when the membranes are ruptured and examinations by the vagina or attempts at delivery have been made.

2. When the low cæsarean is decided upon the uterus should be eventrated, the abdominal wall closed, and precautions taken to prevent infection of the peritoneal cavity by "spill," gloves, or instruments.

3. With the uterus eventrated the operation is performed with remarkable ease. Thus its applicability is extended to include the field of the general surgeon by whom most cæsarean sections are performed.

4. The slight increase in shock caused by lifting the uterus out of the abdominal cavity is more than compensated for by the effectual protection afforded to the peritoneum.

REFERENCES

1. Beck, A. C. A description of the two-flap low incision cæsarean section. Surg., Gynec. & Obst., 1921, xxxiii, 290–296.
2. De Lee, J. B. The newer methods of cæsarean section. J. Am. M. Ass., 1919, xxiii, 91–95.
3. De Lee, J. B. and Cornell, E. L. Low cervical cæsarean section; results in 145 cases. J. Am. M. Ass., 1922, lxxix, 115–117.
4. Frank. Ueber den subkutanean Symphysenschnitt. Monatschr. f. Geburtsh. u. Gynaek., 1910, xxxii, 680.
5. Holland, E. Rupture of the cæsarean section scar in subsequent pregnancy or labor. Lancet, 1920, ii, 591; Proc. Roy. Soc. Med., London, Sect. Obst. & Gynec., 1920, xiv, 22.
6. Kerr, Munro. The lower uterine segment in conservative cæsarean section. J. Obst. & Gynæc., Brit. Emp., 1922, xxviii, 475.
7. King, E. L. The end-results of abdominal cæsarean section. J. Am. M. Ass., 1922, lxxix, 112.
8. Kroenig. The transperitoneal cervical cæsarean section of Kroenig. Doederlein's op. Gynaek., 1912, p. 879.
9. Polak, J. O. The present status of operative obstetrics referring to the abuse of cæsarean section. Surg., Gynec. & Obst., 1922, xxxiv, 567.

A ROUND LIGAMENT OPERATION FOR THE SURGICAL CURE OF UTERINE DISPLACEMENT IN SELECTED CASES

By GEORGE L. CARRINGTON, A.M., M.D., Durham, North Carolina

DESPITE the large number of operations that have been devised for the surgical cure of uterine displacement, the pelvic surgeon occasionally is conscious of a lack of satisfaction with the methods at his command. The procedures now commonly employed are concerned with round ligament shortening of some kind, as these in the main have seemed safest and have been attended with best results. The most popular of these are probably the well known Coffey, Simpson, and Baldy-Webster operations. There are, however, a great number of other procedures, many of them perhaps equally widely known and bearing as distinguished names. Indeed, Chalfant, in an excellent article published in 1916, mentioned 99 varieties or variations of round ligament shortening employed by as many surgeons, the object of the operations being the restoring of the displaced uterus to its normal position. In the hands of competent surgeons good results have followed the use of the procedures now commonly used.

There is, however, an occasional patient in whom the round ligaments are so attenuated that there remain only a few strands of muscle fiber covered by a fold of peritoneum. When the surgeon opens the abdomen of such a patient he feels that there is need for something more than can be attained by the usual operations. The condition is not very frequent, but on Dr. Anspach's service at the Bryn Mawr Hospital there were three such patients during a short period of time. In treating the third patient I rather stumbled upon a procedure that seems to me to offer some help in this type of case.

Patient, F. J., was a colored female, age 28, married. She complained of dragging pains in the back; and "womb falls." Patient began to have trouble about 2 years ago. She was pregnant, and noticed that the uterus would descend to the vulval outlet. She was kept in bed throughout most of the pregnancy. Since then she has had backaches and dragging pains.

Her general health has always been good. She has been married 9 years, and has had 5 pregnancies, the fourth resulting in a miscarriage but the others in children who are now living and well.

Physical examination: General physical and laboratory examinations show a well developed patient whose general condition is good. Pelvic examination reveals no discharge, an everted cervical mucosa, relaxed outlet, cystocele, rectocele, descensus uteri, marked retro-flexio-version. Patient desired to have more children.

The patient was operated upon June 26, 1923, at which time dilatation and curettage, trachelorrhaphy, anterior and posterior colporrhaphy, appendectomy, and suspension were performed. The uterus was in a position of extreme retro-flexio-version, and the round ligaments were quite long and small, consisting of a fold of peritoneum over the slightest strand of muscle. It was decided to perform a Simpson suspension, but in the attempt to bring one of the round ligaments extraperitoneally to the position for suture to the rectus sheath, the ligament was torn in two. The procedure employed then was to ligate the round ligament on each side of the tear, and to use the free end of the proximal half of the divided ligament for suture to the rectus sheath, just as the loop of ligament would have been fixed in the Simpson operation. The distal half of the ligament was then carried over the proximal half and sutured to the anterior surface of the uterus, and the two halves sutured together where they were parallel and in close proximity to one another.

To perform the first part of the operation it will be seen that the procedure after division of the round ligament is practically identical with that

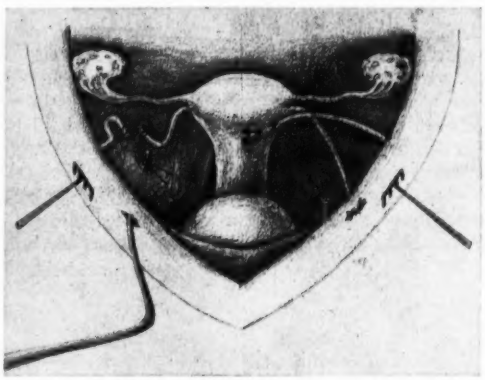


Fig. 1. The right round ligament has been divided and ligated. An Anspach round ligament needle has been passed through a small incision in the rectus sheath and is just emerging through the incision in the peritoneum of the anterior surface of the broad ligament, preparatory to drawing through the free end of the proximal half of the round ligament for suture to the rectus sheath. The completed operation is shown on left side; the proximal half of the ligament being sutured to the rectus sheath and the distal half sutured to the anterior surface of the uterus.

in the Simpson operation. A small incision having been made in the rectus sheath and one in the anterior leaf of the broad ligament, an Anspach round ligament needle or a Kelly clamp is passed through the incision in the rectus sheath, carried extraperitoneally to the little incision in the anterior leaf of the broad ligament, the free end of the proximal half of the divided round ligament grasped and pulled extraperitoneally up to the rectus sheath and sutured to the sheath. The distal portion of the round ligament is then slid over the proximal portion, care being taken not to strip back the broad ligament peritoneum from the round ligament. The free end of the distal half of the round ligament is then sutured to the anterior surface of the uterus and the broad ligament peritoneum attached to it also sutured to the uterus. The proximal and distal halves of the round ligament are then sutured together for the distance that they lie parallel and in close proximity to each other. The procedure can be varied by perforating the broad ligament and carrying the distal portion of the round ligament through the perforation and then suturing it to the posterior surface of the uterus, thus performing a sling operation somewhat similar to the Baldy-Webster, combining, as it were, the Simpson and Baldy-Webster operations instead of the Simpson and Coffey as in the present case. The same procedure, of course, is carried out on both sides.

The only operation that we have been able to find at all similar to this was described by Pankow in 1912. He divided the round ligaments and sutured the proximal end to the internal ring of the inguinal canal, and then implanted the distal end into the uterus, suturing the two portions together along their course. We do not believe with Pankowthat this typeof operation should ordinarily be employed, but we do believe that a double support of the uterus would be worth while in patients with long, greatly attenuated round ligaments associated with marked uterine displacement. We are inclined to believe that the operation we have described would give somewhat better support than that described by him, inasmuch as the rectus sheath gives a firmer anchorage for the round ligament than does the internal ring of the inguinal canal.

There is some question as to the relative importance of the peritoneal and muscular supports of the uterus. Coffey has thought that in his operation the support was principally by the broad ligaments and that the round ligaments later shortened up after the strain had been removed from them. Goldspohn, on the other hand, objected to Coffey's statement that peritoneal folds are the true support of abdominal organs. He maintained that rest would not strengthen muscle and that it would not strengthen the round ligaments. Thus far the question remains unsettled, so that until the truth is determined the safest plan, probably, is to utilize all elements of strength obtainable in difficult cases. Repair of the floor of the pelvis and shortening of the utero-sacral ligaments are often important factors. But we believe that the procedure that we have described above will also be of use in patients who have very thin, long ligaments, since it affords a double round ligament support and a double peritoneal support on each side, thus giving to the uterus practically the combined support of a Simpson and a Coffey suspension.

BIBLIOGRAPHY

1. Chalfant, Sidney A. Present status of round ligament shortening as a surgical cure in uterine displacement. Abstracted, Internat. Abst. Surg., 1916, Nov., 433–445.
2. Coffey, R. C. The principles on which the success of surgical treatments of retrodisplacements of the uterus depends. Surg., Gynec. & Obst., 1908, vii, 383.
3. Goldspohn, A. Some old fallacies in retroversion surgery revived. Am. J. Obst., 1908, lviii, 1035.
4. Pankow, O. Die Behandlung der Retroversio-flexio uteri durch Verdoppelung der Ligamenta rotunda. Zentralbl. f. Gynaek., 1912, xxvi, 1267.

OBLIQUE GASTRODUODENOSTOMY IN THE TREATMENT OF ULCERS OF DUODENUM AND STOMACH AND CANCER OF PYLORUS

By A. L. SORESI, M.D., New York City
Visiting Surgeon, Greenpoint Hospital, Brooklyn, New York

GASTRODUODENOSTOMY is the ideal reconstruction of the passageway between stomach and intestine after resection of the pyloric region. It is ideal because it preserves the physiological relations between stomach and intestine and causes less trauma and shock than does any other procedure.

On account of technical difficulties in its execution and the postoperative complications following its use this ideal procedure is not often resorted to in the surgical treatment of ulcers located in the duodenum or in the stomach near the pylorus or in cancer of the pylorus. We shall not dwell on these points, which are familiar to all surgeons doing gastro-intestinal surgery, but wish simply to present an improved technique which renders gastroduodenostomy more easy of execution and more free from postoperative complications than the procedures generally adopted.

The main points of the technique are:

1. It is not necessary that the opening in the stomach be narrowed in order to be made approximately of the size of the opening of the duodenum previous to the making of the anastomosis.

2. The duodenum is sutured longitudinally to the posterior border of the stomach and then split open along the suture line.

3. The opening between the stomach and duodenum is not narrowed: on the contrary, *at the point of anastomosis* it is wider than in normal conditions.

4. The blood supply along the line of suture is excellent.

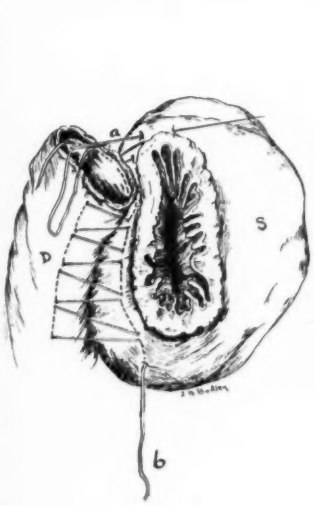


Fig. 1.

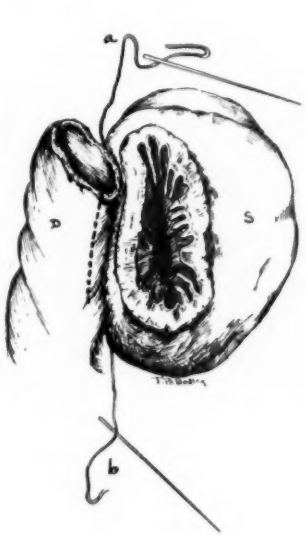


Fig. 2.

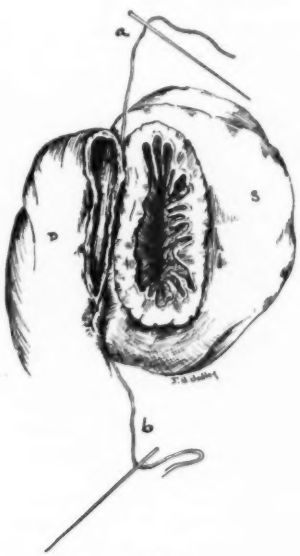


Fig. 3.

Fig. 1. Special mattress suture approximating serosa of posterior wall of stomach, *S*, to portion of serosa of anterior wall of duodenum and to portion of its cut edge, *D*. The suture begins and ends on the stomach. *No knots are made.*

Fig. 2. Mattress suture pulled taut, approximating serosas of stomach and duodenum. The two ends of the thread *a.b.* are held taut by assistant in order to exteriorize the portion of stomach and duodenum which are going to be sutured together. Dotted line shows direction and height of incision on the duodenum proportionate to opening on the stomach.

Fig. 3. The appearance of the stomach and duodenum when the duodenum is incised along dotted line shown in Figure 2.

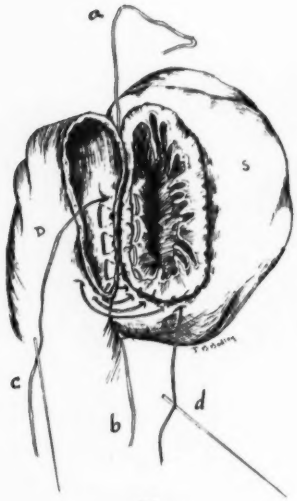


Fig. 4.

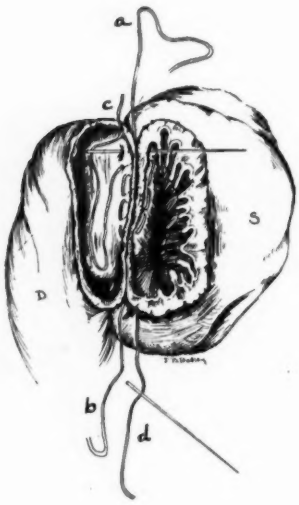
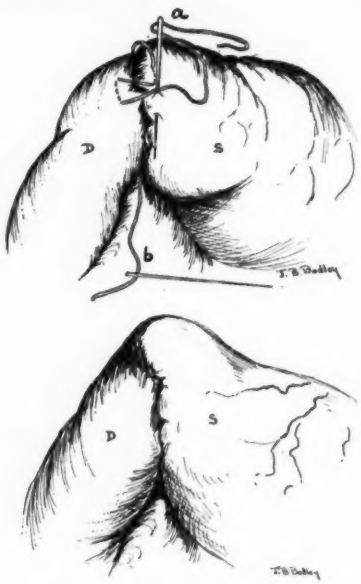


Fig. 5.



Figs. 6 (above) and 7 (below).

Fig. 4. Special mattress deep hæmostatic suture. Note that suture is started on the middle of the posterior cut edges, two needles being threaded to a strand of catgut, and how the lower angle is easily closed without changing direction of the suture and without placing any knot. (The needles from thread *a.b.* have been removed in order to simplify the picture.)

Fig. 5. Lower angle closed by pulling taut suture made by needle *d*. Suture is continued toward the upper angle with needle *c*. and continued all around until it meets point sewn by needle *d*. Note that only one knot has been necessary.

Fig. 6. Deep suture finished and tied. The seroserous suture is continued with needle *a*, until it meets the point sewn by needle *b*. Note that only one knot has been necessary.

Fig. 7. Special shape taken by stomach and duodenum when anastomosed by the oblique method. This shape favors later the formation of a new antrum pylori of normal size.

5. The serosas are broadly approximated by the special double mattress suture used.

6. The disadvantages of the end-to-end anastomosis are abolished but its advantages are retained.

We proceed as follows: After the diseased area of the duodenum or stomach or both is resected, the posterior wall of the stomach is sutured to the anterior wall and to a portion of the opening of the duodenum at about 2 or 3 millimeters from the cut edges (Fig. 1).

The suture on the duodenum should be made the same length as the opening of the stomach. This suture corresponds to the deep posterior suture used in gastro-enterostomy. The two ends of the suture *are not tied*, they are pulled gently until all suture material between stomach and duodenum disappears, and are held taut by the assistant (Fig. 2). The duodenum is cut, as shown in dotted line of Figure 2, parallel to the line of suture and at about 3 millimeters from it (Fig. 3).

The hæmostatic or through-and-through suture is started *in the middle* of the posterior cut edges, two needles being threaded to a strand of catgut. The suture can be started with either needle and directed toward either the upper or lower angles. (In Fig. 4 it is directed toward the lower angle.) When the angle is completely closed, the other needle sutures the cut edges toward the other angle (Fig. 5) and suturing is continued until the last stitch made by the other needle is met. One needle should end the suture on the stomach and the other on the duodenum, at points opposite one another. The two ends of the thread are tied and cut short. Either one of the two needles threaded to the catgut used for the posterior row of seroserous suture is picked up and the seroserous suture is continued about half way (Fig. 6) when the other needle is picked up and the suture is finished. The rents in the gastrohepatic and in the great omentum are closed and the operation is finished.

Special considerations: No clamps are used. A retention suture may be applied to the stomach and duodenum, or they may be held with towel forceps until the posterior row of the seroserous suture is finished. Clamps are not only not necessary, they are dangerous. Clamps traumatize the cut edges, are cumbersome, may render very difficult or impossible the making of an anastomosis that can be accomplished very easily if they are not used. The main objection to the use of clamps is that the cut edges are prevented from bleeding during the time consumed in making the deep or hæmostatic suture. The clamps are removed only when this deep or hæmostatic suture is finished and the opening closed and the cut edges out of view, consequently, *when the surgeon is not sure of the hæmostasis.* He is sure of his hæmostasis instead, if his suture *has stopped all bleeding, while bleeding actually occurred, that is during the time he has applied the deep or hæmostatic suture.*

The thread used for the posterior row of the seroserous suture is held taut and exteriorizes the stomach and the duodenum: it is ideal for this purpose. Leakage into the abdominal cavity is prevented by washing the stomach prior to operation, and properly packing the abdomen. Surgeons will be surprised to see how easily and satisfactorily the stomach and duodenum are exteriorized and leakage is prevented by lifting the stomach and duodenum with the thread used for the posterior row of the seroserous suture.

Objections that may be raised against the procedure are:

1. *The oblique gastroduodenostomy requires more tissue than the end-to-end.* Gastroduodenostomy is only indicated when the stomach and the duodenum can be approximated without tension. The oblique gastroduodenostomy takes up so little more tissue per se that practically in all cases in which an end-to-end anastomosis could be made, also an oblique anastomosis is possible. We feel that the dangers inherent to an end-to-end gastroduodenostomy should lead the surgeon to perform a gastrojejunostomy whenever the stump of the stomach cannot be approximated without tension to the duodenum, as required for a safe oblique gastroduodenostomy.

2. *The opening between the stomach and the duodenum is too large, in fact much larger than the normal pyloric opening.* The width of the opening must be considered at two different periods, namely: the first few days after operation; any time, 1 month or 50 years, after the operation. The first few days after the operation the cut edges of the stomach and of the duodenum become œdematous and are so swollen that the lumen often becomes occluded, thus preventing the passage of the stomach contents into the duodenum. We may add that one of the chief objections to gastroduodenostomy is the occlusion of the lumen by the swollen cut edges. This occlusion causes stagnation in the stomach of anything administered by mouth, dilatation of the stomach, making it impossible to administer not only food but even water by mouth. Consequently in the first few days after the operation, there cannot be any objection to the presence of a large passageway between stomach and duodenum; on the contrary, the larger this passageway, the better.

Later, let us say 1 month or 50 years after the operation, the lumen becomes smaller in all cases on account of cicatricial contraction and proper functions. If the duodenum has been anastomosed to the stomach end-to-end, its lumen might, and too often does, become so narrow that we have the same condition as was met in occlusion of the pylorus due to any pathological cause. If instead, the lumen at the point of anastomosis is sufficiently wider than the normal, cicatricial contraction will not, in all probability cause any dangerous narrowing. Another factor, however, militates in favor of a lumen larger than the normal pyloric opening. The peculiar shape (Fig. 7) taken by the stomach and duodenum at the line of anastomosis does not allow an abnormally rapid emptying of the stomach. This peculiar shape favors a later final improvement of the passageway between the stomach and the duodenum, improvement due to the well known fact that the function makes the organ. After several months we have observed that in the experimental animals the lumen had narrowed to almost normal, the emptying time of the stomach was normal, because a new antrum pylori had formed. This newly formed antrum pylori has the same shape and perhaps the same function as the normal antrum pylori.

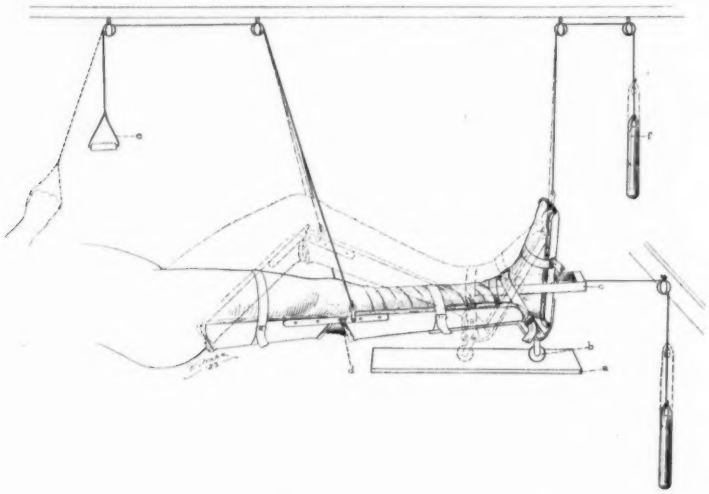
CONCLUSIONS

The author feels that the results obtained in experimental and clinical work justify the statement that the oblique gastroduodenostomy is the safest, most rational, most physiological procedure for the reconstruction of the passageway between stomach and intestine after gastrectomy: oblique gastroduodenostomy, therefore, should be preferred to any other procedure whenever conditions warrant its use. Oblique gastroduodenostomy is the ideal procedure for the surgical treatment of large ulcers interesting the duodenum and the pyloric region but not for extensive carcinomata of the pyloric region.

AN ARTHROPLASTY SPLINT

By PAUL N. JEPSON, M.D., Rochester, Minnesota
Section on Orthopedic Surgery, Mayo Clinic

IT has been the practice at the Mayo Clinic to start motion in the joint that has been operated on in about 5 days, or as soon as the blood clot has become organized. The splint illustrated here has proved to be very satisfactory after arthroplasty of the hip or knee. Immediately after the operation, a Buck's extension is applied to the leg, and then a light cast or a posterior gutter splint. At the end of 5 days this is removed, and the arthroplasty splint, made of aluminum with a lock joint, *d*, to prevent hyperextension at the knee, is applied. To the upright footpiece is fastened a rope running over pulleys on the overhead frame, and to the rope is attached a 6-pound weight, *f*. This part of the apparatus prevents rotation of the leg on the thigh, or rotation of the thigh in the hip. The roller, *b*, beneath the foot-piece is to prevent friction on the bed clothing and operates on a short board, *a*, placed on the bed. The Buck's extension, *c*, is placed on the outside of the foot-piece and permits continuous traction on the knee or hip. Straps are applied to the splint to hold it firmly to the extremity.



Ropes are strung as shown in the diagram, and a hand-hold, *e*, is rigged so that the patient can, by pulling on it, move the knee-joint as often and as much as can be tolerated. After arthroplasty of the hip a special abduction Bradford frame is used in order to afford sufficient bed surface on which to operate the splint. An overhead elevating device is preferable, since it eliminates all unnecessary discomfort caused by moving the patient.

BLOOD TRANSFUSION TO DATE[1]

By EDWARD A. HERR, A.B., M.D., Waterbury, Connecticut
Attending Gynecologist, St. Mary's Hospital

IT is doubtful if the development of any particular phase of surgery has occasioned more ups and downs, more periods of elation and discouragement, more obstacles to surmount and more drawbacks to contend with before success is attained than has modern blood transfusion. Considering in detail the history from its infancy, one is deeply impressed by the interesting sequence of events which has terminated in the present-day achievements.

Reference was made as early as 1492 (35) to the first transfusion as being given to Pope Innocent VIII by a Jewish doctor, three boys being used as donors. All donors died and the patient was not saved. It is doubtful whether reported transfusions at this early date involved more than the giving of blood as a beverage, as the theory of the circulation was not proposed by Harvey (5) until 124 years later (1616).

In 1864 attempts were made to find an innocuous anticoagulant (27). Sodium phosphate and sodium bicarbonate were used but found toxic in doses large enough to prevent coagulation. It remained for Murphy (32) 1897, to give the first effective impulse to surgery of the blood vessels by his end-to-end anastomosis. Up to this time the methods were crude and often attended by fatal results, not more than 50 per cent being successful (5). In France at one time transfusion was prohibited by law.

In 1899–1900 the greatest achievement in the history of transfusion was made when the Englishman, Shattock (39) and the Austrian, Landsteiner (24) simultaneously discovered what was called iso-hæmagglutination, i.e., that serum of one individual frequently agglutinates the corpuscles of another individual's blood. This work was put on a firm basis when in 1906 Jansky (21) and in 1910 Moss (31) classified the blood in four groups and thereby made it possible to select a suitable donor and avoid the disaster of hæmolysis with some degree of certainty. When Crile (9) in 1907 improved on Quierolo's (13) glass tube method (1895) by using a cannula and performing an intima-to-intima anastomosis of the artery and vein and this in turn was improved by the Carrel suture; transfusion might be said to have been established.

Because of the accessibility of the veins Dorrance and Ginsburg (12) advocated the vein-to-vein method as being easier than the artery-to-vein. Janeway (20) further improved the end-to-end method, but on account of its drawbacks this method never became practical. From this time on many workers entered the field to devise methods of simplifying the operation.

It remained for Agote (13) of Buenos Aires and Lewisohn of New York, when they published the results of their studies and experiences with the sodium citrate method in January, 1915, to open up the larger field of blood transfusion. It is, however, to Lewisohn that the profession in this country owes its present knowledge of the citrate method. It is from this method that most of the knowledge of the present-day transfusion has been accumulated and its use made possible on an unlimited scale.

METHODS

Methods of transfusion are classified into (A) modified and (B) unmodified; the latter into (1) direct and (2) indirect. In the modified form the blood is withdrawn, diluted with an anticoagulant, and then injected into the patient. Many reagents have been tried in turn, sodium citrate most successfully. Hirudin is more toxic and the same may be said of sodium phosphate and sodium bicarbonate. Recently Norton (33) of Savannah has used sodium iodide successfully by the syringe method. Some work has been done with sodium sulphate but for some unknown reasons not enough from which to draw conclusions. This anticoagulent is worthy of further development.

Of the unmodified forms the direct, or as it is better known, vein-to-vein or artery-to-vein method, has already been mentioned. This method has proved so cumbersome, so inaccurate and tedious, to say nothing of the skill required in performing it that it has fallen into disuse everywhere except at St. Vincent's Hospital, Richmond, Virginia (18) where it is still used.

A large group of workers unconvinced that the citrate method was adequate and believing in the whole blood theory but wishing to avoid difficulties of the end-to-end method continued their work which has crystallized into the following indirect methods of utilizing unmodified whole blood: (A) the syringe method of Lindemann; (B) the paraffin tubes of Percy, Vincent, and Kimpton-Brown; and (C) an apparatus with the two-way or four-way stop-cock, as the Miller (30) and Unger types.

[1] Read before the Waterbury Medical Association, February 12, 1925.

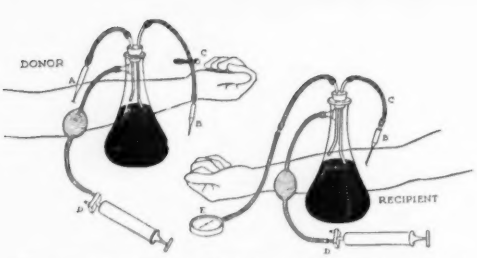


Fig. 1. The Hoffman-Habein Method. Needle *A* is inserted into vein of donor. By the action of the reversible syringe *D* a vacuum is formed in the flask. When sufficient blood has been obtained, needle *A* is withdrawn from vein submerged in sodium citrate solution until the proper dilution has been made. Needle *A* is now exchanged for pressure gauge *E*; air is forced into flask by reversing syringe *D*. Needle *B* is inserted into vein of recipient, clamp *C* removed and transfusion completed under air-pressure.

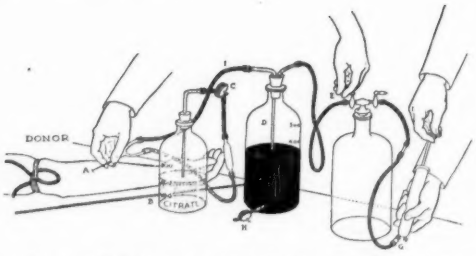


Fig. 2. Brines Method. Needle *A* is inserted into donor's vein. By the action of syringe *G* a vacuum is formed in flask; blood is drawn into flask *D* from needle *A* at the same time with sodium citrate solution from flask *B*. The proper amount of citrate solution is under control by clamp *C*. A clamp is now placed on tubing at *I*; tubing and needle to recipient connected at *H*; syringe reversed at *G*, air pressure caused in flask *F* and controlled by valve *E* and transfusion performed.

In the Lewisohn technique the very simple apparatus necessary recommends it for use by any general practitioner of average skill. The blood when obtained is mixed with 10 cubic centimeters of a 1.5 per cent sodium citrate solution to every 90 cubic centimeters of blood and injected by means of a gravity infusion container. Its advantages are simple technique, ease of performance, and possibility of transporting blood from a donor to a recipient at a distance. Its disadvantages consist of clotting of blood in needles, difficulty of obtaining a uniform mixture of citrate solution and blood, the frequent formation of clots in the receiver, increased likelihood of infection due to exposure to air, and the necessity of filtering the blood to avoid clots. The last adds to the tediousness of operation and the citrate solution destroys the blood platelets which are necessary for maintaining the blood at its highest efficiency.

To overcome the disadvantages of this method many devices have been used. The two most successful are those of Hoffman and Habein (17) and of Brines (5).

Hoffman and Habein use an Erlenmeyer glass flask with a side vent connected with a reversible pump by means of which a vacuum is first formed in the flask and at a later stage in the operation the vacuum is changed to increased air pressure. Through a rubber stopper in the mouth of the flask pass two glass tubes, one connected by rubber tubing to the needle in the donor and the other connected in the same way with the needle in the recipient. A vacuum is formed in the flask by the pump which aids in drawing the blood from the donor after which the needle is withdrawn from the vein in the donor and the proper amount of sodium citrate solution is drawn into the flask by the same needle. The donor's needle is now replaced by a manometer and air is pumped into the flask. The second glass tube through a rubber stopper in the mouth of the flask is now connected with the needle in the recipient's vein and blood properly mixed is pumped into the vein of the recipient.

In the Brines apparatus, three bottles are connected in such a manner that a vacuum formed by a reversible pump in one bottle will draw mixed blood into a second bottle simultaneously from the vein of the donor and a third bottle of sodium citrate solution. The bottle containing the mixed blood is now inverted, held in a standard and connected with a vein of the recipient and air pumped into the bottle to facilitate the flow of the blood into the recipient's vein.

Both of these methods avoid contamination with the air, facilitate the mixing of the citrate solution, eliminate the necessity of filtration, and hasten by air pressure the whole procedure. If the sodium citrate method is to be used, either of the two methods just mentioned are preferable to the older method.

The action of sodium citrate on the blood is manifested experimentally in several ways (41): (1) It destroys the blood platelets which play an important part in coagulation. This method would not then be indicated in transfusion to arrest hæmorrhage. (2) Sodium citrate added to whole blood in amounts necessary to prevent coagulation develops anti-complementary properties in the plasma which can be demonstrated by an attempted Wassermann test. If, however, the same proportion of sodium citrate is added to serum or plasma (without red cells), anti-complementary properties cannot be demonstrated. This signifies

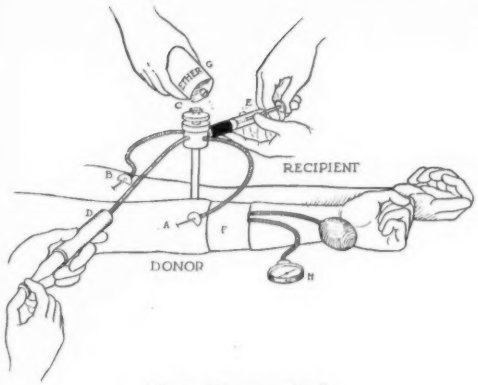


Fig. 3. Unger method.

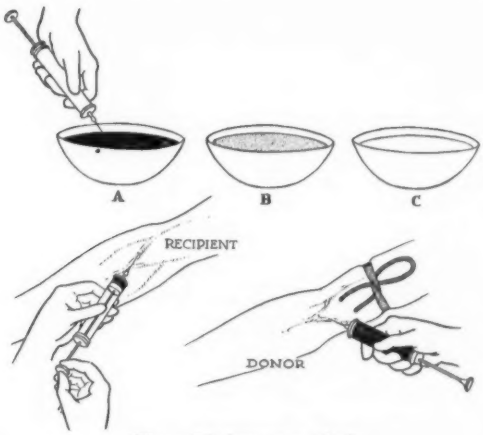


Fig. 4. Lindemann method.

that the salt must alter the red cells liberating in some way anti-complementary substances. Sodium citrate also destroys complement directly and reduces the phagocytic and opsonic powers of the blood. It would therefore not be used in general infections when a resisting power is sought. (3) Sodium citrate increases the fragility of the erythrocytes which is not favorable in anæmias, especially the pernicious type. Tice (40) states that tests for "cell fragility" have a definite clinical value and the fact that the normal erythrocyte yields its hæmoglobin under the influence of a hypotonic sodium chloride solution of a concentration of 0.42 to 0.28 establishes a normal standard of fragility. If now an error is made in the dilution with sodium citrate, the cell fragility is increased just as it is when a tenth normal sodium chloride solution is made up inaccurately.

On the other hand Lewisohn (27) holds that: (1) the citrate method, strange to say, shortens the coagulation time when transfused into the veins but only for a few hours. (2) Recent investigations showed anti-complementary power in citrated plasma but no deleterious effects on the red or white cells. (3) There is a higher percentage of chills following injection by the citrate method than by the Unger but he believes that this is due to chilling of the blood during the transfusion. (4) Eighty-five per cent of the platelets are destroyed and rapidly removed by the spleen, and fresh platelets are mobilized.

In spite of its shortcomings the sodium citrate method has its value under certain conditions and should not be abandoned.

Of the indirect methods, the whole blood transfusion by syringe, the Lindemann method, is the choice of many. This is performed by drawing the blood from the donor by needle into the syringe and transfusing and injecting it into the needle in the recipient. A considerable degree of skill is required to prevent clotting in the needles and the syringes have to be washed in three different basins of saline solution with much loss of time.

Of the paraffin tube methods, the Kimpton-Brown is the most improved. In their technique a graduated glass tube is employed containing a small glass nipple which is inserted into an incision in the vein of the donor and allowed to fill by blood pressure. The tube is then transferred to the recipient and inserted into the vein in a similar manner. A force pump is connected with a vent in the upper part of the tube and the blood is forced into the vein under air pressure. This method utilizes whole blood, avoids contamination, injures the vein by incision, and involves a great deal of skill in lining the tubes with paraffin to prevent clotting. Kimpton (23) states: "I think everybody today agrees that the use of whole blood is the method of choice. I have one patient whom we have subjected to transfusion 40 times, another over 30 times, and several over 20 times. We practically never see a reaction except in those cases in which transfusion has been performed several times."

In the Unger whole blood indirect method the apparatus consists of a swivel or four-way stopcock connected by small caliber rubber tubing on one side to the needle in the vein of the donor and on the other side with a similar tubing leading to the needle in the vein of the recipient. Between the connections in the swivel for these tubings is a hole fitting the nose of a 20 cubic centimeter record syringe by which blood is drawn from the donor. When the swivel is turned toward the recipient

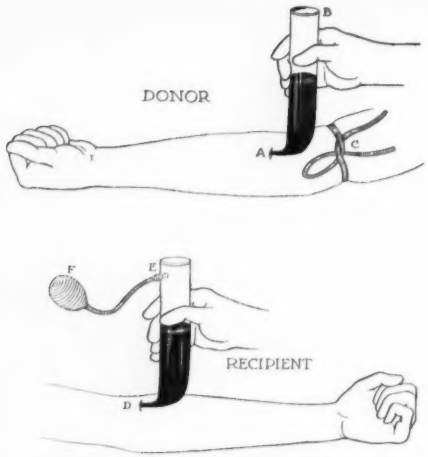


Fig. 5. Kimpton—Brown method.

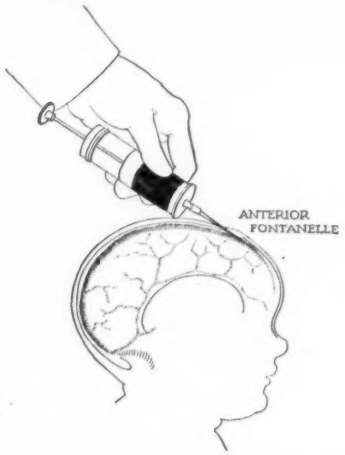


Fig. 6. Transfusions in infants.

blood is forced into the recipient's blood stream. Opposite is another connection for a Luer's syringe of 20 cubic centimeter or 50 cubic centimeter size by which normal saline is forced through the tubings to prevent clotting, always in the opposite direction from that in which the current of blood is traveling. Only a small amount of saline, 2 or 3 cubic centimeters at a time, is necessary to keep the channels clear of blood when not in use. To prevent overheating of the blood in the record syringe a spray of ether is constantly played upon the record and this prevents the formation of all clots. For the actual process of transfusion 10 minutes are required for 500 cubic centimeters of blood and 20 minutes for 1,000 cubic centimeters. Important precautions to be taken are the following: sharp needles should always be used; the veins should be kept well distended by proper pressure; the whole apparatus must be kept cold, otherwise clots will form by heat; and the swivel should be well oiled with sterile olive oil or vaseline.

The ideal method of transfusion should meet the following requirements: (a) whole blood should be used; (b) the blood should not come in contact with the air; (c) only a small corps should be necessary; (d) the transfusion should be performed with speed; (e) a vessel should not be opened except in extreme cases; (f) the apparatus should be equipped to measure the blood; and (g) it should be possible to perform the operation at the bedside. As to the last it might be said that at St. Mary's Hospital we have found it much more convenient to perform transfusion at the bedside in the ward by the Unger method than by the citrate method. All the above requirements are met with in the Unger method. By this method the platelets are not destroyed; the coagulation time is permanently shortened (hence its value in hæmorrhage); if the blood is properly typed there are no deleterious anti-complements formed in the blood and consequently posttransfusion chills are much less frequently encountered.

BLOOD GROUPING

As a routine, a Wassermann test should be done only in exceptional cases where time is an all-important factor. But in all cases the blood grouping must be done before proceeding with the transfusion. Of the two methods, Jansky's and Moss's, the latter is in most common use in this country and is the method we have used in typing all cases before transfusion. This is based on the presence in the blood of iso-hæmagglutinins of which there are two, A and B, providing for 4 possibilities or groups: the presence of both, or of either one, or of none at all.

	SERUMS			
CORPUSCLES	Group 1	Group 2	Group 3	Group 4
Group 1.....	o	+	+	+
Group 2.....	o	o	+	+
Group 3.....	o	+	o	+
Group 4.....	o	o	o	o

From this table[1] it can be seen that if we have 2 known serums, Group 2 and Group 3, any un-

[1] The plus sign indicates agglutination. The corpuscles of Group 1 are agglutinated by the serum of all other groups, but its serum has no agglutinating properties. The serums of Groups 2 and 3 mutually agglutinate each other's corpuscles. Group 4 serum agglutinates all other corpuscles, while its own corpuscles are not agglutinated by any serum. As the essential factor in a donor is the reaction of the corpuscles, Group 4 is the universal donor, Group 1 the universal recipient.

known blood corpuscle group can be ascertained. The technique for obtaining the stock solution is as follows: Blood is withdrawn from 2 known cases of Group 2 and Group 3. The blood is allowed to settle or is centrifuged, the blood plasma is then pipetted off and preserved in the ice box and is usable for 6 weeks as stock serum. This procedure can be simplified by the use of the readily obtainable serums which are on the market. When a donor is to be typed, blood is withdrawn from the ear, centrifuged, the serum, is pipetted off and the corpuscles washed with saline solution. The donor's corpuscles are now matched with a drop each of the known serums of Group 2 and Group 3 and the reaction noted. If there is agglutination in both it is a Group 1 donor. If agglutination occurs with the known Group 3 serum it is a Group 2. If agglutination occurs with the Group 2 it is a Group 3 donor and if there is no agglutination at all, it is a Group 4. Thus it will be seen that Group 4 is the universal donor. When possible a donor of the same group as recipient is selected but a Group 4 may be used if the same group is not obtainable (38). The Mayo Clinic (1) has shown that in their series of transfusions for pernicious anæmia, patients of Groups 1, 2, and 3 were more benefited by Group 4 donors than by their own types.

This grouping is further checked up by matching the donor's corpuscles with the recipient's serum and the donor's serum with the recipient's corpuscles. It must be remembered that the important factor is that the recipient's serum should not agglutinate the donor's corpuscles. We have on several occasions at St. Mary's Hospital, when perfect mating donors were not obtainable, used donors whose serum showed agglutination with the recipient's corpuscles without untoward but with beneficial results following transfusion.

Immediate shock is to be expected only if the blood of the recipient is highly hæmolytic for the erythrocytes of the donor. Under such circumstances destruction of the erythrocytes may follow immediately (22). If the serum is only weakly hæmolytic and the blood grouping poorly defined, hæmolysis will occur late, often too late for a preliminary test (a trial injection of 10 centimeters and observation of the reaction for 10 minutes) to be of use.

When the serum of the blood injected destroys the erythrocytes of the recipient, hæmolysis does not occur before an hour. The clinical picture is determined by the strength of the hæmolytic substances. Depending upon this factor, there may be shock with hæmoglobinuria or merely a variation in temperature, possibly with icterus.

The technique of blood grouping is based on the phenomenon that iso-hæmagglutination occurs independently of hæmolysis and that hæmolysis with few exceptions precedes or accompanies agglutination.

The active life of the red cell is estimated at from 7 to 30 days (40) and is very susceptible to chemical agents and change in the salt content of the blood plasma. Hæmolysis is the robbing of the hæmoglobin content of the cell with active pathological blood destruction and the release of toxic products in the blood stream. It is the effect on some of the component parts of the cell (8).

Bechold (3) recently advanced the theory in explanation of hæmolysis in which he regards the red cell as consisting of a stroma of protein network with a skin of emulsified lecithin and cholesterin across the meshes. He regards hæmolysis as occurring when one of these three component parts is removed.

Iso-hæmagglutination or clumping of the cells is dependent on both the cells and the serum. It affects the cell as an entity in contrast to hæmolysis which affects some of the component parts of the cell. Iso-agglutinins appear in the blood serum of 97 per cent of adults while iso-hæmolysins occur in only 25 per cent.

Recent investigation by Dyke, Oxon, and Budge shows that the agglutinative properties, A and B, can never appear in the offspring without having been present in at least one of the parents, and when inherited, these properties appear in the offspring in accordance with recognized mendelian laws (4). "In considering the group to which the offspring of any two parents may belong, it must be remembered that it is not the group that is inherited. The dominants, A and B, and the recessives, a and b, are the inheritable factors, and it is on the presence or absence of these that the blood group depends." Furthermore in all races whatever the numbers of persons belonging to Groups 2 and 3, those belonging to Group 1 are always the least numerous. Apparently there is some factor that inhibits the ready production of Group 1 and no such inhibition on the parts of the other groups.

That blood groups are inherited and not changed during life was demonstrated by Brines (5) who repeatedly attempted to change the type of an individual by giving repeated transfusions of blood of a different group, hoping to cause accumulation of antibodies in the recipient; but the attempts were unsuccessful.

Familial relationship has a definite relationship bearing on blood groups. Ottenberg (34) after testing a new series of families, confirms the heredi-

tary nature of human groups and suggests its medico-legal application for the detection of parentage. Buchanan (6) holds that Ottenberg's application of blood groups for the determination of the legitimacy of offspring is dangerous. Further work on this subject is necessary (19).

Bauer (2) from his extensive work on 233 hæmophilic families, so far known, gives us the interesting conclusions on hæmophilic heredity. In hæmophilia the sexes are reciprocal; the males are the bleeders, but do not transmit the condition while the females who transmit the condition, do not bleed. In his opinion, the hæmophilia factor is coupled with the sex factor and is a recessive lethal factor.

According to the theory of the biology of heredity, all transmissible qualities are found, from the first nucleus division, in the chromosome constituents of all other cells of the organism, each body cell inheriting the entire original chromosome combination. Accordingly the hæmophilia factor is present in every cell of the body.

REACTIONS

Following blood transfusion, reactions are not infrequently met with and may require combative measures. A reaction may vary from one of slight significance to one of alarming proportions and may appear during the process of transfusion or it may be delayed for a period of 24 hours. In the very mild form in which the patient may experience a sensation of chilliness of a few moments' duration and may exhibit a rise in temperature of 1 degree, no counter measures are required. If the reaction is of a major character with a rise of temperature of 3 degrees and a chill from 10 to 30 minutes in duration, stimulating and supportive measures should at once be instituted.

Reactions are primarily due to error in the laboratory in the typing of the blood. This is avoided by frequently testing out the stock serums used so as to avoid the use of one that has deteriorated by age; and by performing the typing at room temperature, 37 degrees C., as below this temperature typing is inaccurate. With careful and accurate typing in our laboratories reactions from incompatability now rarely occur except in a few isolated para-groups.

In the citrate method the chilling of the blood during the transfer from donor to recipient is given by Lewisohn (27) as more frequently the cause of reactions than any other factor. It is difficult to overcome this obstacle.

The chemical reaction of the sodium citrate salt itself upon the blood platelets is perhaps the largest factor producing the reaction. As the blood platelets are destroyed, toxic by-products are liberated into the plasma with their deleterious after-effects. Mellon (29) has shown that the hydrogen-ion in sodium citrate differs in different specimens of that salt, and it is doubtful whether unvarying solutions of that salt can be produced. In our experience with the sodium citrate method a high percentage of reactions have followed the transfusions.

Again certain reactions are due to the chemical action of the blood on the rubber tubing (7) used during the transfusion. This is easily overcome in the Unger method by using for each operation new tubing which has been boiled in sodium chloride solution for 30 minutes.

Reactions also occur when transfusions are performed within 24 hours after the patient has received ether anæsthesia (26); after the ingestion of certain articles of diet that increase the protein sensitization of the blood; or after bacterins, serums, antitoxins, and coagulants have been used for septicæmia, toxæmia, diphtheria, and hæmorrhage.

INDICATIONS

The field for blood transfusion which originally seemed limited to hæmorrhage and great loss of blood, now that its vast benefits are recognized, has come to include many surgical conditions and a variety of purely medical diseases.

We are all familiar with the case that has been "bled white," is exsanguinated, pulseless, and at death's door showing a brilliant result following transfusion by a glow in the facial color, a return of the pulse, and a restoration of the life. Death in hæmorrhage is due to the starvation of the tissue cells from the lack of oxygen. When now we throw millions of new oxygen carriers in the form of red cells into the blood stream we not only restore the volume but at once inject the indispensable oxygen into the tissue cells. In severe hæmorrhage, when 1,000 to 2,000 cubic centimeters of blood has been lost, 1,000 cubic centimeters or in extreme cases by the use of several donors at the same transfusion 2,000 cubic centimeters may be used. Halbertsma (16) found that to increase the blood count by one million red cells per millimeter it is necessary to transfuse 15 cubic centimeters of blood per kilogram of body weight. Transfusion should be done in all cases with a hæmoglobin under 40 per cent and a red count under 2,000,000 and greater benefit is derived if the red count is even higher. Such hæmorrhages may result from uterine origin, gastric or duodenal ulcers, pulmonary factors, nephritic and bladder hæmorrhages, ruptured ectopic pregnancies, postoperative bleeding, and loss of blood from trauma

as in industrial injuries. Most of the transfusions we have done have been for uterine hæmorrhage, two were for hæmorrhage from duodenal ulcer.

In shock, whether of postoperative or other surgical nature, transfusion is of great value in restoring blood pressure and the oxygen carrying content of the blood. In the Woman's Hospital, New York, donors are typed and held in waiting in cases where postoperative shock is anticipated. Low blood pressure is the indication for transfusion in postoperative shock.

As an anticoagulant to arrest bleeding in hæmorrhage it is unexcelled. In one of our cases of hæmorrhage from duodenal ulcer in which profuse bleeding through the intestinal tract continued for 8 days, calcium lactate was given for 2 days, 3 intravenous injections of thromboblastin were administered on the 2 following days, on the next 2 days two intravenous injections of normal horse serum were given all without effect as the stools continued to show clots of blood for 2 more days and the patient was exsanguinated. A transfusion was followed by immediate control and no more bleeding occurred for 1 month when the patient was operated upon. The arrest of the hæmorrhage is due to the production of new thromboblastic material from donor to recipient.

In pernicious anæmia transfusion replaces the erythrocytes whose destruction exceeds their production. It increases the blood volume which in most cases is reduced (10). It furthermore stimulates the hæmatopoietic organs as is demonstrated by the increase of polymorphonuclear neutrophiles, platelets, and reticulated reds following transfusion (37).

In septicæmia it has not given gratifying results unless, as Unger suggests, the donor is first immunized with the organism obtained from the patient's blood (42). Under these circumstances he reports 5 recoveries out of 7 cases. We recently used transfusion in a case of induced septic abortion in which the temperature was 104 and the pulse 146. In 24 hours the temperature dropped to 99 and the pulse to 100 and complete recovery followed. In another case in which operation was performed for acute osteomyelitis, 2 days after the operation the patient was in hopeless condition with temperature of 105 and pulse 150. Transfusion was done but the case terminated fatally 12 hours later. Our experience with mercurochrome in septicæmia of uterine origin has been no better than with transfusion but we have had excellent results from the intravenous use of tenth normal saline solution.

In obstetrics there are many indications (43) for the use of transfusion including pre-operative preparation, placenta prævia, and melæna neonatorum (44).

It is valuable in anæmia of tuberculosis and nephritis, and in gas poisoning (11) when accompanied by venous section. In anuria and uræmia repeated venous section with repeated transfusion has proved successful (4) and the transfusion is just as important a factor in stimulating the flow of urine in the kidney as decapsulation. In pregnancy with threatened abortion from severe acidosis, transfusion from the donor who has been alkalinized by repeated doses of sodium bicarbonate (15) has prevented the abortion and preserved the pregnancy.

In infants with malnutrition and infantile atrophy, transfusion has saved many otherwise hopeless cases. In the extremely young where the arm veins are inaccessible the transfusion may be made into the femoral, jugular, or the superior longitudinal sinus (25). In (a) sepsis from superficial burns, (b) erysipelas of the newborn, (c) acute septic scarlet fever, and (d) acute intestinal intoxication (36), exsanguination transfusion has reduced the mortality rate to startlingly lower figures. By this method septic blood is withdrawn from the patient (usually an infant) until the point of exsanguination has been reached and then blood equal to the amount withdrawn is transfused.

Other uses for transfusion as in penumonia, typhoid fever, etc., are dealt with in the literature.

Our experience with transfusion of blood has been equally divided between Lewisohn's citrate and the Unger whole blood methods in cases of hæmorrhage from the uterus, hæmorrhage from duodenal and gastric ulcers, osteomyelitis, postoperative shock and in anæmias, cachexia, and sepsis following induced abortions. From our experience and studies we have come to the following conclusions.

SUMMARY

1. Whole blood should be used in transfusions.
2. The Unger method is displacing the citrate method and is the method of choice.
3. Sodium citrate is destructive to the blood platelets and increases the fragility of the erythrocytes.
4. Blood groups cannot be changed during life. The agglutinable properties, A and B, cannot appear in the blood of the offspring without having been present in the blood of the parents.
5. Blood grouping should be done at room temperature (37 C.) and stock serum frequently tested to avoid deterioration.
6. A donor whose serum agglutinates the corpuscles of the recipient can be used and is not

contra-indicated if a donor of complete mating properties is not conveniently obtainable.

7. Transfusions should be used less as a last resort and more as an early therapeutic measure and in a greater variety of cases.

BIBLIOGRAPHY

1. Ashby, W. Transfused blood; Periodicity in eliminative activity shown by organism. J. Exper. M., 1921, xxxiv, 127. Transfused blood. Blood destruction in pernicious anemia. J. Exper. M., 1921, xxxiv, 147.
2. Bauer, K. H. The inherited and constitutional pathology of hæmophilia. Ztschr. f. Chir., 1922, clxxvi, 109.
3. Bechold, H. Bau der roten Blutkoerperchen und Haemolyse. Muenchen. med. Wchnschr., 1921, lxviii, 127.
4. Bowers, C. A., and Trattner, H. R. Repeated venesection-blood transfusion in anuria; Report of a case of acute nephritis with anuria. Surg., Gynec. & Obst., 1924, xxxix, 229.
5. Brines, O. A. Transfusion of unmodified blood. Arch. Surg., 1923, vii, 306.
6. Buchanan, J. A. Medicolegal application of blood groups. J. Am. M. Ass., 1922, lxxviii, 89; 1922, lxxix, 180.
7. Busman, G. J. Rubber tubing as a factor in the reaction of blood transfusion. J. Lab. & Clin. Med., 1920, v, 693.
8. Copher, W. F. Blood transfusion; a study of two hundred and forty-five cases. Arch. Surg., 1923, vii, 125.
9. Crile, G. W. The technique of direct transfusion of blood. Ann. Surg., 1907, xlvi, 329.
10. Denny, G. P. Blood volume in pernicious anemia, Arch. Int. Med., 1921, xxvii, 38.
11. Dorrance, G. M. Indications for blood transfusion. Am. J. M. Sc., 1917, cliv, 216.
12. Dorrance, G. M., and Ginsberg, N. Vein-to-vein transfusion. Pennsylvania M. J., 1912, January.
13. Dutton, W. F. Intravenous therapy. F. A. Davis Co., 1924.
14. Dyke, S. C., Oxon, D. P. H., and Budge, C. H. On the inheritance of the possible existence of a lethal factor. Proc. Roy. Soc. Med., Lond., 1923, xvi, Sect. Path., 35.
15. Gettler, A. C., and Lindeman, E. A new method of acidosis therapy. Blood-transfusion from an alkalinized donor. J. Am. M. Ass., 1917, lxviii, 594.
16. Halbertsma, T. Concerning the quantity of blood administered in blood transfusion. Nederl. Tijdschr. v. Geneesk., 1922, lxvi, 1272.
17. Hoffman and Habein. Transfusion of citrated blood. J. Am. M. Ass. 1921, lxxvi, 358.
18. Horsley, J. S., Vaughn, W. T., and Dodson, A. I. Direct transfusion of blood. Arch. Surg., 1922, v, 301–313.
19. Is parentage determinable by blood tests? Editorial. J. Am. M. Ass. 1922, lxxix, 1246.
20. Janeway, H. H. An improved device for transfusion. Ann. Surg., 1911, liii, 720.
21. Jansky. Haematologische Studien bei Psychiatikern. Sborn. klin., 1906–1907, viii, 85.
22. Jantzen, W. The intravital course of hæmolysis with discussion of blood transfusion and the development of shock from transfusion. Klin. Wchnschr., 1923, ii, 129.
23. Kimpton, J. R. A personal communication to the author.
24. Landsteiner, C. Zur Kenntniss der antifermentativen lytischen, etc. Zentralbl. f. Bakteriol. u. Parasitenk., 1900, xxvii, 361. Ueber Agglutinationserscheinungen normalen menschlichen Blutes. Wien. klin. Wchnschr., 1901, p. 1132.
25. Leebron, J. D. A preliminary report on blood transfusion in malnutrition and infantile atrophy. New York. M. J. & Med. Rec., 1923, cxvii, 298.
26. Levine, E. C., and Segal, H. N. Post-transfusion reactions: alterations in the blood transfusion. Nederl. Tijdschr. v. Geneesk., 1922, lxvi, 1272.
27. Lewisohn, R. Citrate method of blood transfusion after ten years. Boston M. & S. J., 1924, cxc, 733.
28. Lindeman, E. Total blood volume in pernicious anæmia. J. Am. M. Ass., 1918, lxx, 1292.
29. Mellon, R. R., Slagle, E. A., and Acre, S. F. Practical application of "Buffers" in regulation of hydrogen ion concentration in intravenous solutions. J. Am. M. Ass., 1922, lxxviii, 1026.
30. Miller, G. I. Blood-transfusion. Long Island M. J., 1916, 189.
31. Moss. Studies in iso-agglutinins. Bull. Johns Hopkins Hosp., 1910, xxi, 63.
32. Murphy, J. B. Resection of arteries and veins injured in continuity end-to-end suture. Experimental and Clinical Research. Med. Rec., 1897, li, 73–88.
33. Norton, W. A. New and rapid method of blood transfusion. Surg., Gynec. & Obst., 1924, xlii, July.
34. Ottenberg, Reuben. Medicolegal application of human blood grouping. J. Am. M. Ass., 1922, lix, 2137.
35. Pemberton, J. deJ. Blood transfusion. Collected Papers, Mayo Clinic, 1918, x, 508.
36. Robertson, S. B. Exsanguination transfusion: a new therapeutic measure in the treatment of severe toxæmia. Arch. Surg., 1924, ix, i.
37. Roessingh, M. J. Test of bone marrow function. Deutsche Arch. f. Med., 1922, cxxxviii, 367.
38. Sanford, A. H. Selection of the donor for transfusion. Surg., Gynec. & Obst., 1918, March.
39. Shattock. Chromacyte clumping in acute pneumonia and certain other diseases and the significance of the puffy coat in shed blood. J. Path. & Bacteriol., 1900, vi, 303.
40. Tice, F. Practice of Medicine. W. F. Prior Co., Inc., vol. vi, 706.
41. Unger, L. J. Precautions necessary in selection of donor for transfusion. J. Am. M. Ass., 1921, lxxvi, 9.
42. Idem. The transfusion of blood from immunized donors. Laryngoscope, 1923, xxxiii, 145.
43. Williamson, H. C. The use of blood transfusion in obstetrics and gynecology. Am. J. Obst. & Gynec., 1920, i, 188.
44. Woltmann, H. Transfusion by the citrate method in a sixty-hour-old baby with melæna neonatorum. J. Am. M. Ass., 1915, lxv, 2163.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

FRANKLIN H. MARTIN, M.D. ..... Managing Editor
ALLEN B. KANAVEL, M.D. ..... Associate Editor

WILLIAM J. MAYO, M.D. ..... Chief of Editorial Staff

OCTOBER, 1925

USE OF LUGOL'S SOLUTION IN EXOPHTHALMIC GOITER

PLUMMER in 1922 advocated the use of Lugol's solution in exophthalmic goiter to quiet down the storm of acute hyperthyroidism and prepare for operation.

It was but a few years ago that the administration of iodine to an acutely sick exophthalmic goiter patient was strongly warned against, because it was thought that the disease was due to an excess of the normal product of the thyroid in the body, and it was known that this product contained iodine. The use of iodine as a skin disinfectant and of iodized catgut was given up in many clinics. Plummer noticed, however, that the administration of a dose of thyroxin to an exophthalmic goiter patient produced no exaggeration of the symptoms, and because of this, he reasoned that some of the typical symptoms of exophthalmic goiter, at least, must be due to an abnormal product of the gland lacking iodine. He started to administer Lugol's solution on a very definite theory; that exophthalmic goiter is due to an intensive stimulation of the thyroid, under which the gland delivers not only an excess of its normal product, but an abnormal product which is an incompletely iodized thyroxin molecule; and that the symptom complex of exophthalmic goiter varies with the relative amount of the normal and abnormal uniodized thyroxin molecule, the latter giving rise to the characteristic nervous and eye phenomena of exophthalmic goiter which Lugol's solution has been found to control so well. If the thyroid is well trained to this intensive stimulation, the product of the gland is essentially normal—though excessive, and postoperative deaths are rare in spite of the high basal metabolic readings. If the thyroid is not well trained, the stimulation produces an excess of the abnormal secretion, and postoperative deaths are common and are due to the reaction that occurs in these patients overloaded with this abnormal product.

It is most astonishing to observe the change that takes place in the condition of the acutely sick exophthalmic goiter patient after the administration of Lugol's solution for 6 or 7 days; to witness the improvement in the nervous system of the patient as the pulse and metabolic rate fall. It is not unusual to prepare patients with a metabolic rate of well over plus sixty, and a pulse rate of one hundred and thirty or higher, for a safe thyroidectomy after a week of preliminary preparation with Lugol's solution—all of whom, with the former methods of preparation would have had single or multiple ligations with single or double lobectomies, performed months after the ligation with the morbidity extending over a year in some instances.

By the use of Lugol's solution the need for ligation has been greatly reduced, and the

percentage of ligations has fallen from 50 to 90 per cent in some clinics. In the writer's opinion ligations should not be entirely abandoned, but should be held in reserve as a safe measure for the critically sick patient and for the occasional one who does not respond to the Lugol's preparation.

One frequently sees a case *in extremis* upon admission—deep in the throes of a crisis—improve so much under the iodine preparation, administered by the rectum or by painting large areas of the skin with the tincture of iodine, while the vomiting lasts, that operation may be performed safely at the end of 2 weeks. Before we used the iodine preparation, the treatment of such a case was about as successful as was the treatment of a patient in diabetic coma before we had insulin. When one can operate safely upon such cases now it is extremely difficult not to become enthusiastic over the whole subject!

Just a word of warning. With the general employment of iodine in goiter prophylaxis and with a vague understanding which many have that Lugol's solution will cure goiter, one sees many patients who are harmed by the incorrect use of iodine. It is well known that quiescent adenomatous goiters are made toxic by the use of iodine; the effect of iodine upon the active adenomatous goiter is still a debated question. There are some investigators who think it does harm, others who think it does not. Many colloid goiters in patients over 25 years of age contain small adenomata, and these patients are harmed by the administration of iodine.

The writer has been amazed very often to find the large doses of iodine which are given to patients for long periods of time without further examination or observation of those patients by physicians, who have but little knowledge of goiter pathology. The reluctance which some surgeons have to administer Lugol's solution may be due to the fact that they have witnessed the harm which arises from its incorrect use, or by using it incorrectly themselves.

Lugol's solution given to exophthalmic goiter patients exactly as Plummer advises greatly reduces the mortality and morbidity of the disease. The writer considers it the most valuable contribution of the many which Plummer has made to the study of this obscure and treacherous disease.

DONALD GUTHRIE.

BORDERLINE CASES

The surgeon can judge safely and correctly of the state of his patient only when he is at the same time a physician. Moreover, the physician who refuses to treat surgical patients and attends solely to the treatment of internal diseases, must have some surgical knowledge or he will make the grossest blunders.—Billroth.

WE have been accustomed to speak of certain cases as being on the borderline between medicine and surgery. The divergence must be in the way of treatment, because in the bright lexicon of diagnosis, there are no such words as medical or surgical. As doctors, we must meet on a common ground and exist for a common purpose. We must focus ourselves on the patient and ask ourselves, not how we can parcel him out, but how we can best cure him. It is not all of medicine to write a prescription nor to take the blood count; neither is it all of surgery to cut, nor all of operating to have the patient get well of the operation.

All cases can be divided into three main classes: first, those that are frankly medical, or at least, those which it is necessary and proper for the practitioner of medicine to treat; second, those that are on the fence, in which both medicine and surgery are needed;

and third, those that are obviously surgical either at the beginning or at some time during their course.

1. We refer first to those cases that are considered to be in the province of the medical practitioner, whether he be an internist or one of that lost tribe who attend to the wants of men from door to door. The only real specialist now left is the general practitioner. These diseases are his province: pneumonia, typhoid fever, influenza, meningitis, malaria, the diseases of childhood, and the chronic degenerative ailments.

The complications of these conditions, however, may be surgical at any time, not to be treated necessarily by the specialist, but by the practitioner himself. These complications must be looked upon with the mechanical eye. There are certain conditions with which every internist and every medical man should be familiar, but they are more or less surgical, and must be treated by either manipulative or operative surgery. There are operations which the practitioner should perform, but he must not call this minor surgery. Minor surgery is that branch of the art which is done by the minor surgeon, and the more minor the surgeon, the more major the operation will become before he is through with it. The real surgeon is content to say that he is trying to do surgery. He does not attempt to separate it into degrees. There is no other department of medicine which is so divided. Do we hear of minor neurology, or major ophthalmology? Do we hear of minor medicine and major medicine? Let us be as kind to the surgeon.

There should be a tremendous field for the medical man, the man with the medical mind, in the after-treatment of the surgeon's patients. Most of the best operators are not the best therapeutists. One should have around him his best prepared internist, not alone for the cases that are doing poorly, but also for those that are doing very well, to keep them from doing poorly. He knows something about stimulation, or the avoidance of it, and he will carry the patient along with that insight into his comfort that we do not always possess.

2. There are certain types of cases in our second class which may be designated as those "on the fence." Those are the cases in which both the physician and the surgeon are needed in the closest association. Reference need be made to only a few of these types to illustrate what is meant: cases of involvement of the thyroid body, the stomach and its related organs, the gall bladder, the prostate gland, and the abdomen.

The thyroid gland, the governor of the engine—it is shaped like one—is the enigma of the human body. When we say that all thyroid cases should be submitted to surgery, or that none should be, or that the X-ray or medicine will cure them all, we are certainly far from the truth; but, if we say that all of these agents or methods have their place in certain cases, we are very near it. The recent revival of the administration of iodine in exophthalmic goiter brings us back to the time when it was considered very improper to put such patients on that treatment. Thirty years ago, when some of the older doctors were treating their patients in that way, a number of them got well. We have now come to know that it has actually cured types of this disease, which makes us realize that there is nothing new except what has been forgotten.

The stomach sounds the alarm for the rest of the body, and it is well to remember that, when a patient complains in the region of the stomach, the pathology may not be in the stomach. A sickening sight, a foul smell, shocking news, may first be felt in the gastric region. There are only two real diseases of the stomach, cancer and ulcer, and those are so related to near by and distant pathology that

we should begin our search for stomach conditions somewhere else than in the stomach.

Disease of the prostate gland is not always surgical. There are some cases that do not get to the surgeon and there are some that should not get to him. We remove the prostate gland and sometimes the patient does not get well. We must think of the things back of it all. Those are the things that will sometimes kill the patient with or without removal of the gland and are to be taken thought of by the surgeon as well as by the physician.

All the cases of abdominal ptosis are certainly on the borderline—they are amidships. Whenever we see a surgeon do a hammock operation, we wonder if the patient had not better be put into a hammock on the porch rather than have several hammocks hung inside of her. The visceroptosis may be part of a general derangement, the result of loss of nerve tone or of deficiency in fat; and whenever these things themselves are corrected, we may not worry over the ptosis. We have a right to ask whether the ptosis causes the neurosis or whether the fact that the patient is neurotic is behind the whole affair. We see this condition in patients with a long waist, drooping shoulders, slanting abdomen, and invariably, so far as I have seen, they are of the neurotic type, either the still, the excited, or the depressed variety. At any rate, if we should have this condition ourselves, we might prefer to lift the foot of the bed and sleep that way for several months, take an alkaline bitter tonic, wear an abdominal support, and drink buttermilk and some oily material, rather than submit to multiple plicating operations.

3. The last class, those conditions that are frankly surgical in the beginning or at some time during their course, include appendicitis, gall stones, intestinal obstruction, herniæ, and tumors, both benign and malignant. We all might agree that these represent the common illustrations of conditions which are purely, and perhaps only, remediable by surgery.

A most difficult diagnosis to make is chronic appendicitis. We are frank to say that we shrink from operating for chronic appendicitis unless we can prove that at some time the patient has had a definite attack, watched and supervised by a competent physician. Even then, we might have our doubts. The only thing we can be sure of is to practice the art of exclusion; and then after eliminating every possible cause for the symptoms, perhaps we can be persuaded that the appendix might possibly be removed.

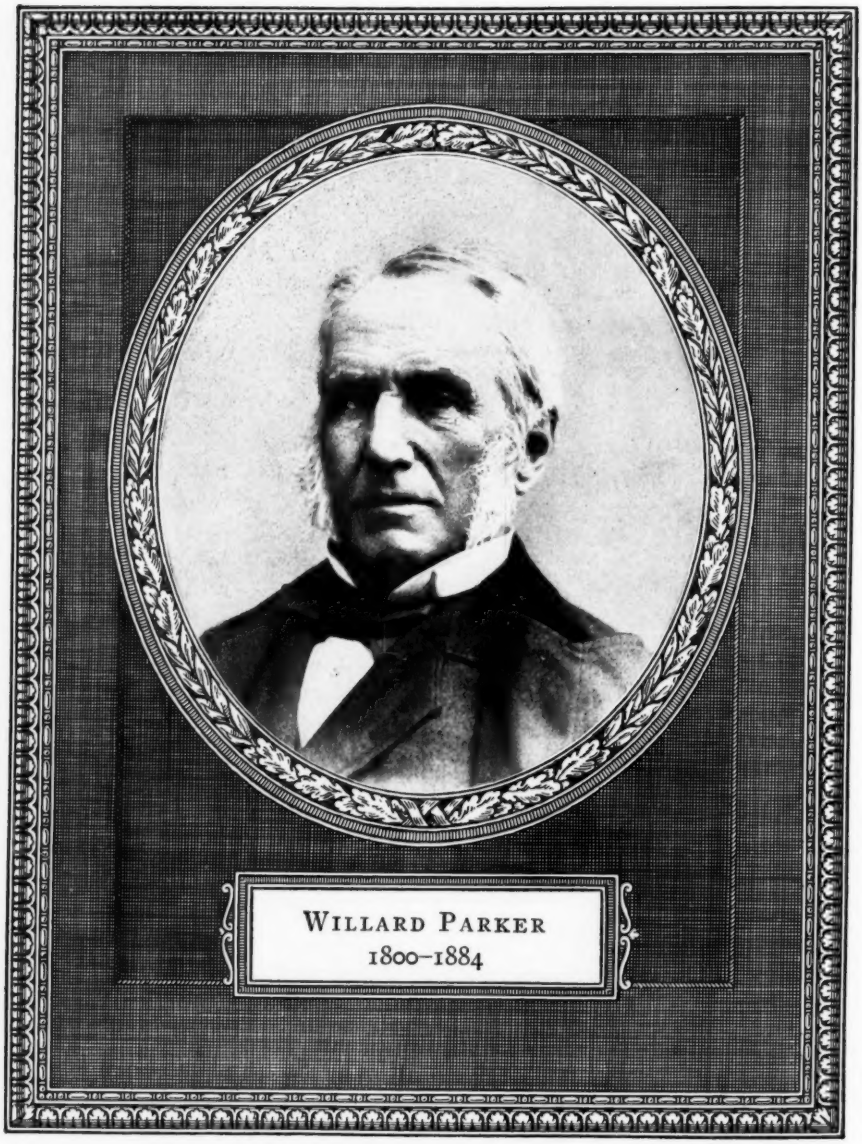
Gall stones, purely an incident in gall-bladder infection, usually have to come out because they produce pain, and sometimes the gall bladder should come out with them and sometimes it should be left in.

Intestinal obstruction, when fæcal impaction is ruled out, is so definitely a surgical disease that comment seems unnecessary.

Hernia is always a surgical subject, whether remedied by mechanical or operative means. We feel sure that every surgeon agrees that trauma is not the original cause, that every case would be better operated upon early in life to prevent all the possibly dangerous sequelæ which may come with age, and that our results usually are good.

The breast may be taken as an example in which both malignant and benign tumors occur, and we may thank the propaganda recently started for the large number of benign cases we are now getting, in spite of the too great prevalence of the well advanced malignant ones. There is only one thing to say, and that is, that every lump in the breast should be removed, and that all growths in the borderland of "possibly malignant" should have a radical operation performed on them, coupled with everything else we can do for the patient.

HUBERT A. ROYSTER.



WILLARD PARKER
1800–1884

MASTER SURGEONS OF AMERICA

WILLARD PARKER

THE nobility of the soul, the loftiness of the ideals, the force of the character and the influences of the life, of an illustrious man of the past, can be vividly portrayed to posterity no more aptly than through the voice of the man's contemporaries. Thus for telling truly and graphically the tale of the distinguished life of Dr. Willard Parker, there have been selected from the memorial address to the latter, delivered before the New York Academy of Medicine by his intimate friend, Dr. William H. Draper,[1] the following passages:

"Nearly six months ago that familiar and honored name, Willard Parker, was blotted from our roll, but in our memories it is so deeply graven that the sound of it will always recall to mind one of the most notable figures in the circle of distinguished men in which he moved for so many years. He filled, perhaps, for a long period, a larger place in popular and professional esteem than any of his contemporaries, not because of his superior genius, nor because of great acquirements, but rather because of a character that somehow grasped at once the affections of his fellow men, and made them trust and honor him.

" . . . and yet there are few perhaps who lived nearer to him than I did for more than thirty years, no one I am sure he inspired with a warmer affection or more exalted regard. . . . I can only crave your indulgence if I seem to exaggerate his virtues or to overestimate the influence which he exerted for more than forty years in this city as a physician and a public teacher.

"The story of Dr. Parker's life is not so remarkable for the incidents or even for the achievements of his career, as it is for the singular power he wielded in his professional relations to his patients, his pupils, and the public through the simple force of his personality.

"He was born with the century, which his life nearly spanned, in the town of Lyndeborough, New Hampshire. . . . He was inspired by his Puritan forefathers with the love of freedom and the dignity of labor. He tilled the soil on his father's farm. . . . He prepared himself for college with the rewards of his own toil, and graduated at Harvard in 1826. His ardent religious nature inclined him to the profession of the ministry, but an incident in his Freshman year happily diverted his thoughts toward a calling in which his mind escaped the fetters of

[1] Tr. M. Soc. New York State, 1885, p. 328.

dogmatic theology, and left his religious enthusiasm free to expend itself in practical Christianity. One of his classmates had a strangulated hernia, the local physician called to his aid the celebrated Dr. John Collins Warren, and young Parker was so powerfully impressed with the sagacity and the skill of the surgeon, who speedily reduced the hernia, that he at once resolved to devote his life to the study and practice of the healing art. . . . Shortly after receiving his degree" (Harvard 1830) "he was appointed Professor of Anatomy in the Berkshire County Medical College at Pittsfield, Massachusetts, at that time one of the leading schools in the country.

"In 1832 he was appointed Professor of Surgery in the Pittsfield school, and for four years he held both chairs, lecturing twice daily. In 1836 he moved to Cincinnati, where he was called to the Professorship of Surgery in the Cincinnati Medical College. He remained there for three years. It was during this period that he visited Europe and spent some months in observing the methods of the foremost surgeons of that time in England and France. In 1839 he was called to fill the chair of Surgery in the College of Physicians and Surgeons in this city, and here for more than thirty years he labored with unflagging zeal as a teacher of the principles and practice of surgery.

"His fame as a brilliant lecturer and an accomplished surgeon, his noble presence and the wonderful charm of his manner, soon achieved for him all the success to which the highest ambition could aspire, and amid all the temptations of personal popularity and pecuniary ease, he never lost his enthusiasm or abated his labors in behalf of the object which was always nearest his heart, the elevation of the standard and the improvement of the methods of medical education.

"In connection with the late James R. Wood, he reorganized the old Almshouse at Bellevue into a Hospital, and served there for many years as one of the attending surgeons. He was appointed an attending surgeon of the New York Hospital in 1856. . . . On the establishment of St. Luke's, the Roosevelt and the Mt. Sinai Hospitals he was made a member of the staff of consulting surgeons in these institutions. Indeed he was so identified with the growth of charitable enterprises, in the way of hospitals and dispensaries in this city, that he was associated with the organization of almost all of them. He was one of the first, and for many years one of the most active members of the Pathological Society and of the Medical and Surgical Society. His deep and abiding interest in this Academy is known to you all. He was one of its honored Presidents.

"He resigned the active duties of his Professorship in 1870, and was made Emeritus Professor of Surgery. . . He was made a Doctor of Laws by Princeton College in 1870.

"He was essentially a broad man with an unbounded faith in the possibilities of the science of healing and an enthusiasm that disappointment never abated and failure could not quench. He could not be called a learned man, but

he was what some learned men never become, a wise man. He acquired his art mainly at the bedside, and it was there that he displayed most conspicuously the qualities which gave him his high claim to distinction as a physician and surgeon. He was always self-possessed, no emergency disconcerted him, no difficulties appalled him. He was uniformly calm and master of the situation. He was a keen and comprehensive observer. . . . He was sagacious in diagnosis. . . .

"No one who has ever seen him enter a sick room can forget the magical influence of his alert and cheerful presence. It was as if he brought with him the talisman of health. It banished fear and inspired hope.

"It was in his character, however, as a public teacher that Dr. Parker impressed himself most powerfully upon all who came within the sphere of his attractions. He loved to teach. . . . There was something about his enthusiasm that was contagious. . . . He was the pioneer in introducing clinical lectures into the college instruction. . . . When he entered the amphitheatre his presence seemed to fill it; he riveted attention. . . . His glance was an inspiration and his voice like the voice of a prophet. His manner toward his patients commanded confidence and assured sympathy.

"He never lost an opportunity to impress upon his pupils the limitations of the cure of disease as contrasted with the ever-widening possibilities of its prevention.

"It is to be regretted that Dr. Parker was not gifted with a faculty for literary work. . . . He was singularly free from prejudices and ever ready to acknowledge that new ideas and new methods might be better than the old.

"Dr. Parker may be said to have originated the operation of cystotomy for irritable bladder," . . . and "the operation for perityphlitic abscess," . . . the latter in 1864 which "it is certain that he was not aware that Mr. Hancock, of London, had done. . . . successfully in 1848.

"He was a man of public spirit. He was interested in all great social questions. The public health was to him a subject of the deepest concern. . . . To him and the late Dr. John O. Stone we owe the reorganization of our Health Board.

"He recognized in the reckless use of alcoholic stimulants one of the chief causes of physical degeneracy, as well as of the poverty and crime, in our times, and he showed by his denunciation of intemperance his exalted conception of the duty of a physician as the conscientious and uncompromising guardian of health. He was for some years the president of the Inebriate Asylum at Binghampton.

"He was generous, truly, to a fault; he was quick to recognize merit and encourage it. He loved to do a kindly act and to speak or write a friendly word. . . . He was conspicuously the friend of young men.

"We are impressed chiefly by his ardent love for his calling, by his entire devotion to its high behests and by the singular purity and nobility of his per-

sonal character. To these he owed his eminent success in his profession, his title to rank high as a physician and teacher, and his acknowledged position in the community as one of its most valued citizens. He dedicated himself to his work with his whole heart and mind and strength. He never wearied in his efforts to augment its usefulness, to maintain its honor and to exalt its claims to public confidence.

"He was always aspiring to a clearer vision, he was free from the fetters of jealousy and conceit, and untrammeled by the clogs of self indulgence. He served his fellow men; he strove to be a lamp unto their feet and a light unto their paths."

Dr. Parker died in 1884. A portrait of him hangs in the Surgeon-General's Library at Washington. The Willard Parker Hospital for contagious diseases of the Health Department of New York City was erected in his memory.

This record of accomplishment and of influence exerted by Dr. Willard Parker, furnishes a striking example of the capacity of a single individual to do good by a well-spent life. Lives like this one, so rich in kindliness and love for one's neighbor coupled with force of character directed for good, can well be kept before the public mind down through the ages, as an inspiration to all in every walk of life.

WILLIAM CHITTENDEN LUSK.

TRANSACTIONS OF SOCIETIES

CHICAGO GYNECOLOGICAL SOCIETY

REGULAR MEETING HELD MAY 15, 1925, DR. CAREY CULBERTSON PRESIDING

SPECIMEN OF CARCINOMA OF THE APPENDIX

DR. W. C. DANFORTH: This is a specimen from a woman 74 years of age whose previous history was of no importance. She consulted her physician because of enlargement of the abdomen and obstinate constipation. A mass was palpable in the abdomen. X-ray showed some impingement on the sigmoid. She was rather thin and the mass could be felt most prominently on the left side. It was suggested that she had a carcinoma of the ovary which was probably not operable. The abdomen was opened and a large mass occupying the left half of the pelvis was found. There were numerous metastases in the abdomen. About the eighth or ninth postoperative day she had a little hypostasis in both lungs and died quite suddenly from rupture of the left ventricle caused by an occlusion of the coronary artery. The specimen presented is a carcinoma of the appendix. There were also large secondary masses in the omentum which were firmly adherent to this mass. This is the second carcinoma of the appendix that I have observed.

The other case was relatively non-malignant. The appendix was found to contain carcinoma but the patient has remained well.

SPECIMEN OF CARCINOMA OF THE APPENDIX

DR. SYDNEY SCHOCHET: This is a specimen of primary carcinoma of the appendix, found on routine examination. The majority of these cases are clinically benign. However, 6 per cent of the cases recorded show metastases. Probably the case which Dr. Danforth reported belonged to this group.

REPORT OF A CASE OF NATAL TEETH

DR. SCHOCHET: The mother was a ii-para, and when the child was born two lower incisors were present. This is a comparatively rare condition. In the literature of the Paris Maternity Hospital during a period of 10 years there were only 3 cases in 10,375 babies.

DR. BAER: This condition is not rare.

HÆMATOSALPINX; RUPTURED CORPUS LUTEUM

DR. J. P. GREENHILL: The two specimens are taken from patients who had symptoms and signs of an ectopic pregnancy but in whom operation revealed pathological conditions much more unusual than those ordinarily found in extra-uterine pregnancy.

One patient had had a full term intra-uterine pregnancy 17 years ago and an ectopic pregnancy in the right tube 12 years ago. Dr. De Lee terminated the tubal pregnancy by a partial salpingectomy. The patient this time came to see Dr. De Lee saying she had an ectopic pregnancy on the left side. She had been bleeding for 27 days. Pelvic examination revealed a tender, doughy mass on the left side. I punctured the cul-de-sac to see if there was any blood in the peritoneal cavity. No blood was found so I performed a posterior colpotomy and made a careful examination. I found on the left side what I thought was a typical unruptured tubal pregnancy. The mass measured about 8 by 4 by 4 centimeters. A laparotomy was performed. The ovary on the right side was normal and to it was attached the preserved proximal end of the right tube. On the left side was the enlarged bluish tube which was felt through the colpotomy incision. The left tube and the left ovary which was cystic were removed. Examination of the tube showed it to be a hæmatosalpinx with no evidence of a pregnancy.

The second specimen was from a patient 34 years old who had 3 living children.

Twenty-seven days after her last menstrual period she had a sudden attack of abdominal pain following which she fainted four times. Two physicians were called both of whom made a diagnosis of acute appendicitis and advised operation which the patient refused. Dr. De Lee examined the patient on admission to the hospital and felt a tender doughy mass on the right side which he diagnosed as an ectopic pregnancy. He referred the patient to me. I made a pelvic puncture and found old blood. I performed a laparotomy and found an unusually large amount of free and clotted blood in the peritoneal cavity. The left adnexa were negative. On the right side was a large blood-clot adherent to the ovary. When this clot was removed there was revealed a large corpus luteum with a long rent in its outer edge. The blood in the peritoneal cavity had come from this tear. The right tube was œdematous and reddened. Both tube and ovary were removed on suspicion of a possible ovarian pregnancy but sections of the ovary showed only a corpus luteum, with a large tear in its surface.

DISCUSSION

DR. MARK GOLDSTINE: Is it a routine practice to make a vaginal puncture in every suspected ectopic pregnancy? If you find blood and you have not an ectopic, of what value is a vaginal puncture?

DR. EMIL REIS: Will Dr. Greenhill tell us something about the history of menstruation in connection with these cases?

DR. C. E. PADDOCK: Do I understand that unless you find blood you do not operate? Undoubtedly a majority of ectopic pregnancies are destroyed early and cause no trouble; but when the diagnosis is doubtful, the abdomen should be opened.

DR. J. P. GREENHILL (closing the discussion): I did the punctures because Dr. De Lee asked me to and because we do pelvic punctures in nearly all the cases where extra-uterine pregnancy is suspected. If old blood is found we operate. If no blood is found we usually wait. In the first case I followed the puncture by a posterior colpotomy, and outlined the tubal mass with my finger in the peritoneal cavity.

In answer to Dr. Reis, the first patient had her last period March 18, when she began to bleed, and bled until she came to the hospital on April 14. Pelvic puncture was done 3 days after admission. The March period came 3 days earlier than it was expected. The patient complained of severe backache, her breasts were enlarged, and she felt she was pregnant but in an abnormal way.

The second patient had her last period on March 23. She had an attack of pain and fainting on April 19. She had no external bleeding whatsoever, and I operated on her the day she came in. The corpus luteum was about 2 centimeters in diameter and corresponded in appearance to what is generally found at the premenstrual phase of the menstrual cycle.

If old blood is found in suspicious cases we always operate. If blood is not found Dr. De Lee usually waits. He has had a number of cases during the last few years in which he has obtained dry taps. He did not operate and according to the subsequent history of these patients nothing developed. We consider a pelvic puncture as we do a laboratory test or X-ray, that is, an additional means of arriving at an accurate diagnosis before operation.

FETAL HEART SOUNDS AS A DIAGNOSTIC AID

DR. LOUIS RUDOLPH: The location and the intensity of the fetal heart sounds in obstetrical diagnosis have not received enough attention. Since Mayor of Geneva described the fetal heart sounds in 1818 and Lejumeau de Kergaradec published his monograph in 1822 very little has been added to our knowledge in the interpretation of the fetal heart sounds.

In pregnancy the fetal ovoid in a known position and presentation may have an associated minor degree of deflexion. A delay in the progress of labor gives the suspicion that the fetal ovoid is complicated with a slight degree of deflexion. When the head is high and the cervix is not sufficiently dilated for palpation of the sutures and fontanelles, the location and the intensity of the fetal heart sounds is an aid in determining the cause of delay in the progress of labor, particularly in these days when rectal examinations are largely used in the conduct of labor.

The variations in the transmission of the fetal heart sounds which are heard externally follow certain laws governing the transmission of sound. The intensity as well as the direction of the sound is dependent upon the conductivity of the media intervening between the cardiac chamber, where the sound is produced, and the external abdominal wall. Irrespective of the presenting part, the fetal body may be in an attitude of flexion or extension. Therefore the fetal heart sounds from the cardium are transmitted to a point on the anterior or posterior fetal thoracic wall. (Fig. 1).

The fetus being in either an attitude of flexion or extension is usually in contact with the uterine wall, and where this point of the uterus comes in contact with the abdominal wall, the point of maximum transmission is located. Therefore the maximal sounds are transmitted from the fetal cardium respectively through the fetal back or chest, the uterine wall, and the abdominal wall. The intensity of the sounds will depend upon the intervening media (intestines and liquor amnii), and the character of the abdominal wall. In the literature the location of the points of maximum intensity of the fetal heart sounds is uniform as shown in Figure 2.

These facts have been accepted and standardized, because in the mechanism of the cephalic and podalic positions the fetal ovoid has been held to be in unvarying attitudes of flexion, except those that are known to be deflexed such as brow or face.

In recent years roentgenography has come into use as a diagnostic aid in obstetrics. These roentgen studies may change our conceptions of the attitude of the fetus in utero during pregnancy and labor. Warnekros has clearly demonstrated by serial roentgen pictures that in the last few months of pregnancy and during labor the attitude of the fetus is of varying degrees of deflexion in known positions and presentations. Previous to the use of the roentgen-ray, many obstetricians by the usual means of abdominal palpation and rectal or vaginal touch, must have felt that the fetus was in some degree of deflexion in known flexion attitudes. I have repeatedly observed, in palpating the fetus in utero late in pregnancy and during labor, that in some cases the fetus is not as flexed as others have been in the same position and presentation.

In viewing roentgenograms, one sees many variations in the relation of the fetal spinal column to the maternal spinal column, and to the maternal sacroiliac synchondrosis. In cephalic positions the fetal spinal column may lie over the maternal spinal column, and then may be placed at different intervals laterally, until it is found deep in the flank, free from the maternal spinal column. If the fetal spinal

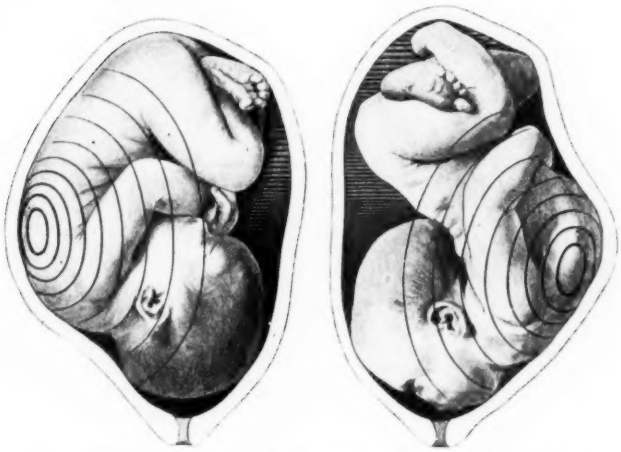


Fig. 1. Transmission of fetal heart sounds in flexion and extension.

column is found lying over the maternal spinal column, the maternal sacro-iliac synchondrosis is not covered well by the fetal head, but as the fetal spinal column is found laterally in different degrees, so is the maternal sacro-iliac synchondrosis covered more by the fetal head, as it becomes more flexed.

The text-books on obstetrics state that the position of the fetus in utero is in a compact attitude in known flexion positions, but not in the known deflexion attitudes.

In the normal anterior and posterior positions the landmarks where the fetal heart sounds are heard are designated on Figure 1, the mode of transmission being by way of the back, the sound waves traveling in circles as shown in Figure 1 (left). In the deflexion attitudes the sounds are heard in the quadrant of the abdomen on the side opposite the back, the mode of transmission being by way of the chest; the sound waves traveling in circles (Figure 1, at right). These known premises demonstrate that the fetal heart sounds are transmitted by way of the back and chest. The questions that arise are: What is the significance of double locations of the fetal heart sounds? What are the modes of transmission? What are the differences in the intensity?

The above considerations of the mechanism of the transmission of the fetal heart sounds raises the question of the significance of double location of the sounds. Warnekros has clearly demonstrated that the fetal ovoid is not in a compact attitude, but assumes an attitude of varying degrees of deflexion of the head and spinal column in the known flexion positions. It is also an established fact that the fetal heart sounds are transmitted by way of the back and chest. Why cannot the fetal ovoid in a flexion position be slightly deflexed *so that the fetal heart sounds are transmitted to the maternal abdomen by way of the back and chest at the same time?* Figure 1 demonstrates the accepted views explaining why the fetal heart sounds are heard at various points on the maternal abdomen by transmission.

Sound is transmitted by waves which travel in circles. If the fetal ovoid is a compact flexed body, the explanation of the production of other locations

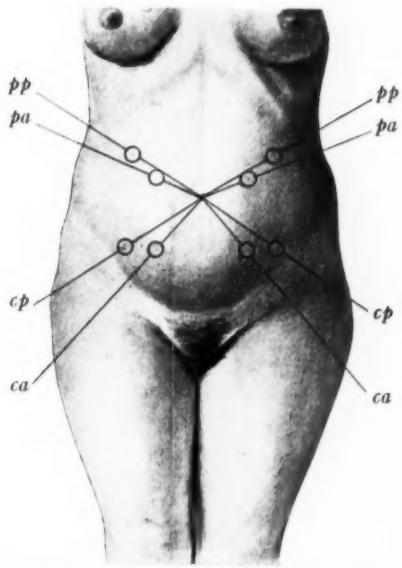


Fig. 2. The location of the points of maximum intensity of the fetal heart sounds is uniform. *pp*, Podalic posterior; *pa*, podalic anterior; *cp*, cephalic posterior; *ca*, cephalic anterior.

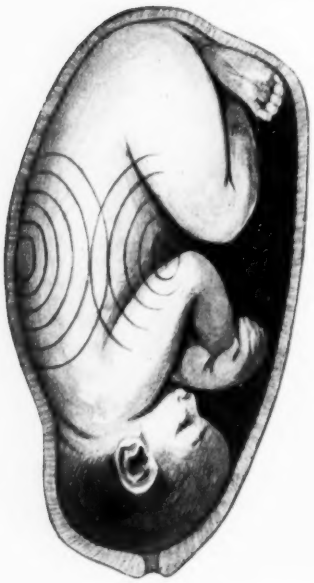


Fig. 3.

Fig. 4. A roentgenogram taken when the cervix was dilated to 4 centimeters, head high.

of the fetal heart sounds is readily understood. When more work is done in obstetrical roentgenology and our knowledge regarding the attitude of the fetus in utero is increased, we will change our opinions of the transmission of the fetal heart sounds. If sound waves travel in circles and if the fetus in many positions of normal flexion attitudes assumes slightly deflexed attitudes the fetal heart sounds may be *transmitted by way of the back and chest at the same time* with the result that the sounds from both sides overlap each other. These sounds being synchronous as to rate and rhythm are difficult to differentiate by auscultation (Fig. 3).

The intensity of the fetal heart sounds is dependent upon the conductivity of the intervening media. If we take into consideration the recent roentgen pictures of the fetal attitude, we can see that in flexion attitudes we have minor degrees of deflexion in pregnancy and labor, and the intensity of the transmitted sound is due *to the conductivity of the intervening media and the attitude of the fetus in utero.*

I have been studying the significance of the fetal heart sounds in areas not related to the back in flexion, or the chest in deflexion attitudes. By palpation and rectal or vaginal examination, I have diagnosed normal cephalic positions and presentations, but suspected some degree of deflexion of the fetal head and spinal column. I have found that I could auscultate fetal heart sounds in other locations and of different intensities. I have observed in some cases of normal flexion presentations that the progress of labor was atypical in normal pelves and that the labor was protracted.

The following case led me to attempt an explanation of the variations of the fetal heart sounds.

Mrs. I. W., age 28, ii-para, came for examination March 14, 1923. Her family and personal history were negative. Her first labor was in 1917 with normal delivery. The last menses were September 10, 1922; labor was expected July 17, 1923. The pelvic measurements were: interspinous 26, intercristal 30, bitrochanteric 32, Baudelocque 20; transverse outlet 10, anteroposterior 11.

On June 18, 1923, a diagnosis of right occipito-anterior was made; the head was high and the fetal heart sounds were heard in two planes, lower right and left quadrants of the abdomen, and were of equal intensity. Prenatal examinations subsequently gave the same findings.

The patient entered the Mount Sinai Hospital on July 16, 1923, at 12:05 p. m. with pains every 10 minutes and weak. The cervix was effaced, but there was no dilatation; the head was high and the bag of waters intact. The fetal heart sounds were heard over the right and left lower quadrants and were of equal intensity. A diagnosis of right occipito-anterior with some deflexion of the fetus was made.

The progress of labor was slow. Examination at 3:30 p. m. showed 1 centimeter dilatation and the head high with pains every 10 minutes and weak.

July 17, at 10:30 a. m., the cervix was dilated to 4 centimeters, head high, and the fetal heart sounds were the same. A roentgenogram was taken (Fig. 4).

At 3:45 p. m. examination showed no further progress. At 4 p. m. the membranes ruptured

spontaneously and in a few minutes the perineum began to bulge. At 4:15 there was complete dilatation with the head on the perineum. The fetal heart sounds were found on the lower right quadrant. Spontaneous delivery occurred at 4:43 p. m.

At about the thirty-sixth week of pregnancy a diagnosis was made of right occipito-anterior and the fetal heart sounds were heard over the lower right and left quadrants of the abdomen. The fetal back did not appear to be deep in the flank. When the patient went into labor, the diagnosis before roentgen examination was right occipito-anterior, with some deflexion in a normal flexion attitude. No vaginal examinations were made throughout the labor; by rectal examination the relation of the fontanelles could not be determined on account of the high head. After the roentgen picture was studied, I felt that my diagnosis was confirmed.

In normal flexion attitudes there are four types of variations in the fetal heart sounds:

1. *Complete flexion.* With the fetal back deep in the flank of the mother, the heart sounds are heard only over the position of the back.

2. *First degree of deflexion.* The fetal heart sounds are heard distinctly over the fetal back, and faintly in the opposite quadrant or in relation to the fetal chest.

3. *Second degree of deflexion.* The fetal heart sounds are heard over the lower right and left quadrants and are of equal intensity, being in relation to the back and chest.

4. *Third degree of deflexion.* The fetal heart sounds are faint over the fetal back and distinct on the opposite quadrant or in relation to the fetal chest.

Routine prenatal care. A diagnosis is made of the position and presentation by palpation and auscultation of the fetal heart sounds in relation to the back and chest at the thirty-sixth week, and the locations and the intensity are recorded. The fetal heart sounds are systematically recorded until the end of labor. I have found cases in which the sounds have passed progressively through the four stages enumerated. I believe it is possible to follow cases from complete flexion attitudes to extreme deflexion (face) attitudes, by following the fetal heart sounds progressively through the stages enumerated and then finding the heart sounds as present in the complete deflexion attitude.

Anterior cephalic presentations. At the thirty-sixth week the sounds are heard only in relation to the back. On subsequent examinations the sounds are found in the same or in some type of the variations. At the thirty-sixth week the sounds are heard in one of the types of the variations. These variations frequently change from the thirty-sixth week to the termination of labor.

Posterior cephalic presentations. At the thirty-sixth week the sounds are heard only in relation to the back, deep in the flank, or according to the types of the deflexion variations. More frequently I have found double location of sounds with varying degrees of intensity. During the pregnancy and labor the intensity changes. In a few cases of left occiput presentations at the thirty-sixth week of pregnancy, I found the fetal heart sounds deep in the left flank, but on subsequent examinations the fetal heart sounds were heard only in the right lower quadrant. These cases gave me grave concern, because I feared right mento-anterior presentations. At delivery they proved to be occiput presentations.

Persistent occiput presentations. When the fetal heart sounds were heard only in relation to the chest the labor was protracted. In a few cases I found that during labor the sounds were heard faintly in relation to the back which was a good prognostic sign that flexion was taking place, and that rotation would therefore be hastened.

Transverse arrest. When the rotation of the head is arrested in the transverse diameter and the sagittal suture palpated, and the relation of the fontanelles determined, I have found in most cases double locations of the fetal heart sounds with varying degrees of intensity.

Podalic presentations are illustrated by the following case: Mrs. B. S. At the thirty-sixth week a diagnosis of sacral left anterior was made; the fetal heart sounds were heard only in the right lower quadrant of the abdomen. This location persisted throughout the pregnancy and labor. At delivery the presentation was sacral left anterior, with legs extended. The explanation for this location is that the placenta was implanted on the anterior surface of the uterine cavity, the placenta was large, which caused deflexion of the back; the fetal chest was displaced downward and outward, in relation to the lower right anterior uterine wall, and the heart sounds were transmitted to the right side of the maternal abdomen.

CONCLUSIONS

1. The fetal heart sounds are not transmitted only from the back or chest to other areas on the abdomen.

2. Frequently sounds from two locations are transmitted to the abdomen at the same time by way of the back and chest.

3. Normal positions and presentations may have an associated slight deflexion of the fetal spinal column.

4. The interpretation of double location and intensity of the fetal heart sounds is an aid in determining the cause in the delay in the progress of labor.

DISCUSSION

DR. W. A. N. DORLAND: This paper is interesting to me especially because of the possibility of variations in the outline of the normal fetal ellipses during labor. The work of Warnekros has beautifully shown this variation in labor. Recently we had an X-ray taken of a breech presentation at 8 months which shows that the fetal ellipse is not fixed during pregnancy. It is easy to see from this picture how it might be difficult to hear the fetal heart sounds unless flexion were complete.

VISCOSITY OF THE BLOOD IN THE PREGNANT

DR. HELIODOR SCHILLER, and DR. AUGUSTA R. FELSHER: The cells of the living body are constantly bathed in serum which furnishes nutrition and the necessary water, and carries away waste products and injurious substances, or promotes their chemical combination with other substances into harmless substances. A constant exchange between the fixed cells of the organs and the circulating cells and serum of the blood takes place.

Metabolism, nutrition, absorption, secretion, excretion between the organ cells and the blood and lymph stream depend on diffusion, osmosis, filtration, and lipoid solubility. As long (Wells) as the cell is living there is no constancy in composition, for metabolic processes cause constantly varying intracellular osmotic conditions; new chemical reactions are set up and new osmotic currents formed. The constant upsetting of the equilibrium is what constitutes cell life.

Of the aforesaid powers, diffusion and filtration depend among many other factors upon the resistance, the inner friction which the blood experiences in circulating through the smallest vessels and capillaries—the resistance to the relative motion of its constituent parts, called viscosity. Viscosity of the blood differs quite markedly in its phenomena from the viscosity of liquids or fluids, because blood is not homogeneous but we have the blood corpuscles and blood platelets suspended in the blood. Blood differs from a liquid not only because it is not homogeneous, but also because blood is a colloid fluid with all the characteristics of the colloids. Viscosity and diffusion are correlated. Not less is viscosity dependent on osmotic pressure, represented by the total amount of electrolytes in the plasma and the non-electrolytes, such as sugar and urea. Oscillations in one or a group of them will reflect upon the other, osmotic irritation is followed by osmotic reaction. The ability of the blood and of the body cells to regulate osmotic pressure is great. The cells regulate pressure by taking in water, by making use of the secretions from the salivary and sweat glands, the stomach, and the intestines (diarrhœa) and principally, however, by increasing or decreasing the amount of urine to reduce or increase osmotic tension.

The protein-salt union is of the greatest importance for the diffusion of the colloids, and since they materially change the viscosity of the blood, they reflect the importance of viscosity upon the metabolism in general. And if we consider the constant change of the blood gases, oxygen and carbon dioxide, with the accompanying change in the blood bicarbonate figures of the plasma, the increase in size of the erythrocytes with increased carbon dioxide tension, the change of the solids in the plasma, the constant changes in the plasma of glucose, uric acid, urea, amino-acids (ingestion), lecithin, creatinin, cholesterol, acetone bodies—to repeat, the ever changing gas tension and changing electrolytes, the wide changes during digestion—we must realize that the viscosity of the blood, interdependent so much on all these factors, is a constantly changing wave. And therefore it is not surprising that we find that viscosity will change very markedly in the same individual at different hours of the day and on different days.

A diet rich in proteins, a vegetarian diet or diet rich in fats, hunger, overeating, alcohol, tobacco, reduced or increased intake of water, rest, labor, increased perspiration, diarrhœa, drugs, different altitudes, all produce changes in viscosity. Viscosity is different in men and women and children, different in the tall and heavy and lean and slender; heavy work increases viscosity—loss of water and increased total protein content of the blood. It is increased in cases of vitium cordis, labor is quickly followed by marked increased viscosity. The differences in viscosity above mentioned, however, are not large, the blood protects tenaciously its protein and salt content and tries to hold it always at the same level. Hess and other investigators found a parallelism between the hæmoglobin content and viscosity, between the specific gravity of the blood and viscosity, and also between blood pressure and viscosity.

Viscosity increases with the protein content, also with the content of mineral salts. However iodine salts seem to make an exception. They lower the viscosity of the blood, which to some extent might explain the good results in hypertension following the use of potassium iodide, even when syphilis is not the underlying cause.

The number of blood cells naturally influences the viscosity as in cases of polycythæmia which show high viscosity. The same situation is present in high altitudes and after a prolonged cool bath. After bleeding the viscosity is low through hydræmia of the blood, the water of the plasma being the first to recuperate its quantity. Transfusion with normal salt solution lowers the viscosity, laked blood has very high viscosity through freeing of highly viscous proteins.

If we hold in mind that viscosity influences diffusion (osmosis) so important for metabolism, that increased viscosity of the blood must necessarily throw correspondingly increased work upon the propelling forces of the blood (the heart and arterial walls) we readily see that clinically, but not diagnostically, viscosity plays an important rôle. Viscosity is not only an expression of the inner friction of the blood flowing in the capillaries, it is a true chemical and biologic picture of the condition of the blood.

The normal viscosity as compared with water under the same conditions ranges in women of the ages between 10 and 20 years from 3.1 to 4.9; between 20 and 30 years, from 3.5 to 5.1. The methods used, the source of blood either direct from the vein or from the ear (peripheral blood), the very important factor of the temperature in the room, and many other details will affect the results. However, if the same observer practices one method to perfection and observes certain rules strictly, for instance per-

forming the experiment at the same time of day with the same room temperature, etc., certain conclusions may be drawn.

The apparatus and method we used for the estimation of viscosity was that of Hess. We made at first a great number of estimations on the normal and not until we had the method mastered did we start our investigation. Peripheral blood was used. The coagulation time was estimated by the capillary pipette method, and controlled by the slide method under the microscope.

Ancient physicians such as Galen claimed that the blood in pregnancy is thicker than normal. It is considerably richer in blood lipoids, cholesterol, cholesterol esters, and lecithin; and fatty acids and the globulins are considerably increased. In the later months of pregnancy we find a lower plasma bicarbonate level, a state of an uncompensated acidosis with increase of ketone bodies. The slight cyanosis present could be produced by an impediment to breathing through the large uterus—over carbonization of the blood—but may be an undersaturation with oxygen produced by an abnormally great reduction of the blood oxygen during the passage through the placenta. If we consider then that at least in the later stages of pregnancy there is an over carbonization, the strongest factor in the increase of viscosity; if we consider the increased lipoids, kidney and liver disturbances with retained nitrogen proteids and urea, which are correct for a good many cases; then we should expect an increased viscosity in pregnancy.

The average viscosity findings of different authors as collected in the literature in 54 women is 4.2 while I found the average viscosity of 30 pregnant women to be 4.63 in the latter part of pregnancy. Case 24 shows the highest viscosity, 6.7; a coagulation time of 1.50; and 1,100,000 platelets. Case 28 shows the lowest viscosity, 2.2, a coagulation time of 5.30, and 170,000 platelets. The blood pressure was 92–72, the hæmoglobin 68; and there were present œdema of the ankles and eyelids, anæmia, and hydræmia which explained to some extent the low viscosity.

The low readings of viscosity we found in a woman pregnant two months; a viscosity of2.05,coagulation time 2.50, hæmoglobin 70, and blood pressure 116–80. In another woman 3 months pregnant the viscosity read 2.75, coagulation time 4.20, hæmoglobin 65, blood platelets 90,000. This woman had two quite severe hæmorrhages and a possibility of a hydatid mole was considered. Thus the average viscosity in the later months of pregnancy is about 0.4 higher than normal.

Surprising are the findings in 15 women about the end of the first week after delivery. The average viscosity, in spite of the recent loss of blood, is very high, 5.17. Case 8, 7 days postpartum has a viscosity of 7.10, a coagulation time of 1.10. All 15 women nursed their babies. The cause of the rather marked increase in viscosity must be looked for partly in the highly viscous protein constituents of the blood during the process of involution of the uterus and partly in lactation. The blood platelets were counted in 10 women, two of them in the second and third months of pregnancy, the rest toward the latter part of pregnancy. The average number of blood platelets in these 10 women was 392,000, the average in the normal women between 250,000 and 500,000. Case 24 shows a high count of 1,100,000, a coagulation time of only 1.50. In Case 46, no platelets could be seen, either in the native or stained slide. Case 47 has a very low count of 90,000.

Concerning the correlation between coagulation time and viscosity, it can be said that there seems to be in some cases a strict parallelism between them. Case 8 shows a viscosity of 7.1 and a coagulation time of 1.10; Case 4 a viscosity of 6.7 and a coagulation time of 1.35; Case 24 a viscosity of 6.7 and a coagulation time of 1.50, blood platelets 1,100,000. But this parallelism does not hold good all the way through my cases. Case 10 with a viscosity of 5.9 has a coagulation time of 3.15; Case 29 a viscosity of 5.8 and a coagulation time of 5.30; Case 27 a viscosity of 4.1 and a coagulation time of 6.10. On the other side low coagulation is correlated to a high blood platelet count and vice versa. However, the number of blood platelet counts is too small to allow definite conclusions. This much can be said:

1. The viscosity of the blood of pregnant women in the latter part of pregnancy is higher than in the non-pregnant.

TABLE I.—AVERAGE VISCOSITY

Case No.	Month of pregnancy	Viscosity	Coagulation time	Age	Child	No. of blood platelets
1	9th	4.50	3.15			.....
2	7th	4.60	3.30	19	2nd	.....
3	8th	4.85	3.30	32	3rd	.....
4	9th	4.80	5.30	19	2nd	.....
5	9th	4.95	2.15	38	4th	.....
6	9th	5.90	3.15	29	5th	.....
7	7th	4.80	3.00	21	1st	.....
8	6th	4.45	4.10	34	2nd	.....
9	6th	4.10	3.45	22	1st	.....
10	8th	5.25	4.00	21	1st	.....
11	5th	4.10	4.30	25	1st	.....
12	6th	4.45	2.50	20	1st	.....
13	8th	4.70	3.30	32	3rd	.....
14	5th	4.25	4.50	20	1st	.....
15	7th	4.55	4.15	36	1st	.....
16	8th	4.10	3.45	22	2nd	.....
17	6th	3.95	7.00	22	6th	.....
18	7th	4.30	4.00	25	1st	.....
19	6th	4.25	3.25	24	1st	.....
20	7th	4.05	3.15	24	2nd	.....
21	6th	4.70	4.45	20	1st	.....
22	6th	3.75	4.30	19	1st	.....
23	7th	4.30	5.00	23	3rd	380,000
24	10th	6.70	1.50	24	4th	1,100,000
25	8th	5.25	2.40			680,000
26	6th	5.45	4.30	26	3rd	800,000
27	3rd	4.10	6.10	36	8th	70,000
28	7th	2.20	5.30			170,000
29	5th	5.8	5.30	27	2nd	180,000
30	9th	4.10	3.45	29	3rd	450,000
31	2nd	2.95	2 50	16	1st	
32	3rd	2.75	4.20	18	1st	90,000

TABLE II.—VISCOSITY ABOUT WEEK POSTPARTUM

Case No.	Day post partum	Viscosity	Coagulation	Age	Para
1	11th	4.85	3.6	38	4
2	10th	4.90	3.15		
3	½	5.10	2.15	36	7
4	10th	6.7	1.35	19	1
5	3rd	4.45	6.	18	1
6	10th	5.50	1.25	18	2
7	6th	5.10	1.50	27	1
8	7th	7.10	1.10	20	1
9	11th	4.65	2.2	30	4
10	10th	5.90	3.15	29	5
11	10th	5.5	1.30	24	1
12	7th	5.9	2.2	27	2
13	9th	5.00	2.30	20	1
14	5th	5.05	4.15	21	1
15	7th	4.05	3.15	24	2

2. It is higher in the second week postpartum in the nursing mother.

3. A parallelism between viscosity and coagulation is not apparent.

DISCUSSION

Dr. C. S. Bacon: This is a very interesting study, but there is no attempt made to make use of this method for practical purposes. For that reason it is all the more interesting. We are generally looking for something that we can use. In the past, attempts have been made to make practical use of the viscosity of the blood but these have failed. It does not follow that the attempts will always fail. My interest in the subject has been to find the relation between the increased viscosity of the blood and increased blood pressure. The relation between the coagulation time and the viscosity is interesting and may have a bearing on the problem.

I arose, not for the purpose of adding anything to the discussion; but I dislike to see a paper that is of such interest as this allowed to pass without a word of comment.

Dr. C. E. Paddock: I agree with the last speaker. It is not right to let this paper go without comment. The essayist has not come to any definite conclusions, but he shows a large amount of work, which, with further investigation, may be of much value.

I would like to ask him if he came to any conclusions as to why the viscosity was higher in the second week of the puerperium than it was at the time of delivery.

Dr. J. P. Greenhill: I would like to ask Dr. Schiller whether any of his patients had toxæmia and if so, whether he found any change in the viscosity of the blood. We know that the blood of toxæmic patients has a tendency to clot very rapidly and although Dr. Schiller said there was no parallelism between blood clotting and viscosity, still there may be a change in the viscosity of the blood of patients with toxæmia due to factors other than abnormal clotting.

Dr. Heliodor Schiller (closing the discussion): Several years ago in studying the blood chemistry of pregnant women I found cholesterol and cholesterol-esters and lipoids very much increased toward the end of pregnancy. I thought this increase easily explained the increase in the viscosity of the blood of the pregnant. This increase of cholesterol and lipoids, however, disappears very soon after delivery, and surely could not offer an explanation for the definite increase of the blood viscosity after delivery. The absorption of products produced by the involution of the uterus might explain it better. All conditions present after delivery would make us expect to find a decrease in viscosity. I have no explanation to offer for the definite increase in the viscosity of the blood which takes place toward the end of the first week of pregnancy.

There were no cases of toxæmia among those investigated. All cases were normal.

TRAITE

DES HERNIES

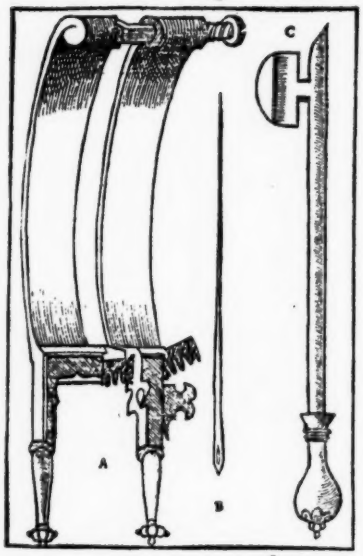
CONTENANT VNE AMPLE declaration de toutes leurs eſpeces, & autres excellentes parties de la Chirurgie, aſſauoir de la PIERRE, des CATARACTES des yeux, & autres maladies, deſquelles comme la cure eſt perilleuſe, auſſi eſt elle de peu d'hommes bien exercee: Auec leurs cauſes, ſignes, accidens, anatomie des parties affectees, & leur entiere guariſon:

Par PIERRE FRANCO de Turvieres en Prouence, demeurant a preſent a Orenge.

A LYON,
PAR THIBAVLD PAYAN,
1561.

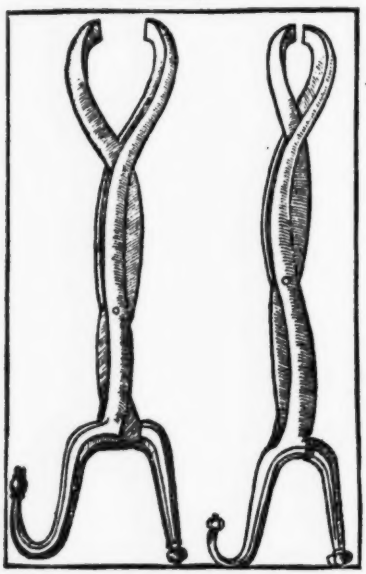
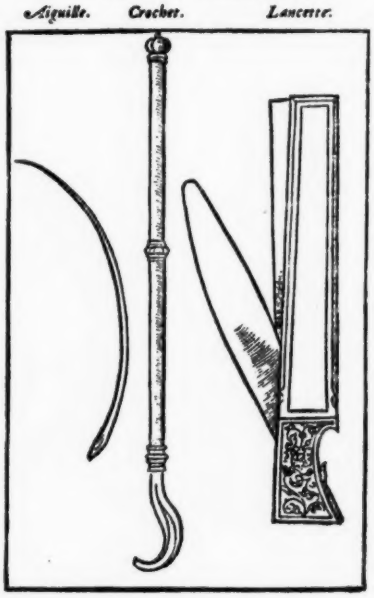
Auec Priuilege pour neuf ans.

A Tenailla aux hernia, B Eguille, C Cautere.



C 2

Tenailla inciſiuæ.



Aiguille. Crochet. Lancette.